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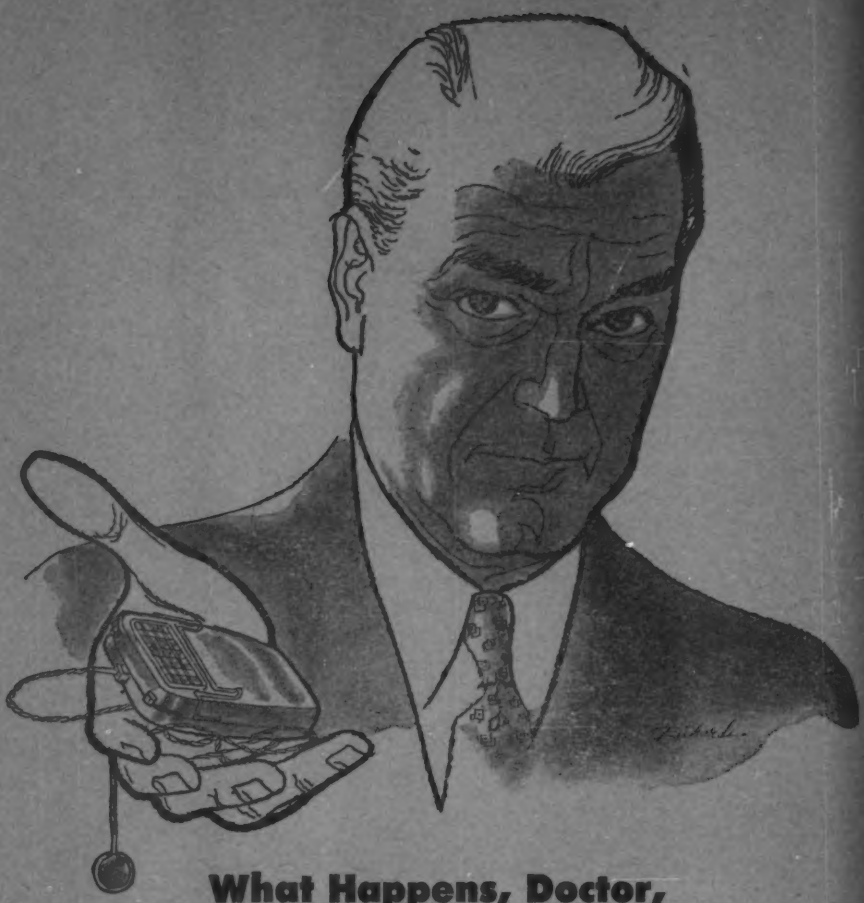

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THE
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VOL. LXIV NOVEMBER, 1954. No. 11

REVIEW OF AVAILABLE LITERATURE ON THE PHARYNX AND PHARYNGEAL SURGERY FOR 1953.*

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ANATOMY AND PHYSIOLOGY.

Bosma[1] states that interest in the motor functions of the pharynx has recently been stimulated by the increasing incidence of bulbar-pharyngeal poliomyelitis. The accumulation of patients having residual difficulties of deglutition and speech, sequelae of this form of poliomyelitis, impelled Bosma to study further the motor mechanisms of the pharynx and to seek new methods of correction of these particular handicaps. In an excellent, well illustrated article, Bosma shows the methods used in the study of cadaveric material as well as the anatomic observations made on living patients whose pharynges had previously been exposed by surgical resection of neoplastic or osteomyelitic tissue in the area of the nose and paranasal sinuses. The anatomy and function of the musculature of the upper pharynx have been carefully studied. The position and action of the levator veli palatini, pharyngopalatinus, salpingopharyngeus and superior constrictor muscles and a number of inter-relations of their action have been identified. The commonly observed action of the pharyngopalatinus, salpingopharyngeus, and superior constrictor muscles was their combined action of elevation and constriction

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of the upper pharynx. The levator veli palatini muscle was identified as a prime mover in this combination of action of the intrinsic muscle of the pharynx.

Palatopharyngeal closure is accomplished by the tensor veli palatine, levator veli palatine, superior constrictor of the pharynx, salpingopharyngeus and pharyngopalatinus. The main function of this muscle valve is occlusion or constriction of the passageway between the oropharynx and the nasopharynx during speech and deglutition. Bloomer[2] has made an extensive study of the movements of these muscles during speech, swallowing, and other mechanisms in two patients with surgical facial defects. Sound and silent movies were made. An excellent record of these observations is presented, which should be of value to the oral surgeon, the prosthodontist and speech pathologist.

Vorhaus and Deyrup[3] describe the accelerating action of ATP on the ciliary movement of the frog's pharyngeal mucosa as "highly consistent, rapid, reversible and demonstrable in the presence of relatively low concentration of ATP." It seems to have a specific effect in that "hydrolysis of the substance abolishes completely its effect on the test system." The mechanism of action of ATP on ciliary movement remains to be clarified and, the authors believe, deserves "careful reinvestigation in the light of recent developments in the field of cell physiology and biochemistry."

PATHOLOGY.

Semenov[4] gives an excellent discussion of the pathology of the pharyngeal recess, Eustachian tubes, tonsils and adenoids from a clinical viewpoint. The pathology of lymphoid tissue is described in detail. The importance of the nasopharyngeal pituitary gland is stressed. The advantages of careful surgical procedures on the adenoid combined with nasopharyngeal radium application over indiscriminate Roentgen-ray therapy to the nasopharynx is stressed. This is an excellent article and should be read in its entirety by all otolaryngologists.

ANOMALIES.

In delivering the Hunterian lecture, Hynes[5] stated that after cleft palate operations speech can be excellent; however, some-

times unsatisfactory results are obtained, usually in patients with an enlarged nasopharynx and a palatopharyngeal space which does not close properly. Hynes, therefore, devised a pharyngoplasty by muscle transplantation to correct the abnormality in these cases. This technique is described and illustrated.

The physiology of the pharynx in relation to voice production is discussed and evaluated. He concluded that in cleft palate in which the result of operation is considered a failure, if the soft palate remnants are reasonable in size and mobility, it is possible to produce a palatopharyngeal sphincter and excellent speech can be expected. If the soft palate remnants are hopelessly small, pharyngoplasty by muscle transplantation should be done.

In a survey of the American literature, McGovern[6] could find no record of the association of congenital choanal atresia with congenital heart disease. In a previous paper McGovern had discussed the physiologic and hereditary aspects of choanal atresia and mentioned associated abnormalities, such as high arching of the palate, asymmetry of the face, bifid uvula, congenital coloboma of the iris, double tragus, double congenital aural fistula, polydactylism and flat nose. In the present article he adds to this list two cases of choanal atresia associated with congenital heart disease, which are presented in detail. As evidence that this congenital anomaly has a hereditary tendency, McGovern cites a case reported by Dixon, of bilateral choanal atresia in twin sisters.

A pharyngeal flap taken from the posterior pharyngeal wall and attached to the posterior surface of the vellum is one of the methods used to lengthen the palate to improve speech. The problem of anesthesia in this limited surgical area is great. Walden and associates[7] have used preliminary tracheotomy and intratracheal anesthesia in eight patients with success.

DIVERTICULA.

Perzik[8] tells us that since 1884 when the surgical treatment of pharyngeal pulsion diverticula was first employed, the vertical incision has been the avenue of approach. This incision has usually extended from the level of the hyoid bone to the

suprasternal notch along the anterior border of the sternomastoid muscle. Perzik is of the opinion that such a vertical incision is a handicap and is limited in the transverse diameter by limited retraction. For this reason he advocates the usual thyroid collar incision, which begins 2 cm. above the clavicle and is carried in a curved line 3 cm. beyond the median line on the right side and 3 cm. beyond the posterior border of the sternomastoid muscle. Illustrations are utilized to demonstrate the new approach, which results in better exposure because of the improvement in the transverse diameter of the field. The reviewers also believe that the collar incision produces a superior cosmetic effect to that of the vertical incision in the neck.

The late Dr. Lahey[9] had the largest published series of surgically treated pharyngoesophageal diverticulas. His mortality rate was the lowest of any large series, consisting in two deaths in 359 patients. Lahey attributed his success in the management of these patients primarily to the use of a two-stage procedure. He gives a lucid description of the surgical technique he employed.

Perzik[10] gives a brief discussion of pharyngoesophageal pulsion diverticula. He describes his one-stage technique for excising the diverticulum using a transverse collar incision.

Phelps[11] presents a discussion of 24 cases of pharyngeal diverticula, 20 of which were treated surgically with no deaths. Serious complications developed in 12 patients. These included fistula, stricture at the site of excision, recurrence, thoracic complications and unilateral vocal cord paralysis. There were no cases of infection. Phelps advises surgical removal of the sac as soon as the diagnosis is made, as the smaller the sac, the easier the performance of the operation. He believes that complications can be prevented by "earlier diagnosis and operation while the sac is small; by better preoperative care; by better surgical technique, including gentle manipulations of the strictures; by putting less pull on the laryngeal nerves; and by accurately suturing the mucosa and muscle layers." He recommends elimination of the feeding tube and intravenous feeding for the first few days.

DISEASES.

Denny and coworkers[12] compared the effects of penicillin, aureomycin and terramycin on acute streptococcal pharyngitis and tonsillitis in a well controlled series, without the benefit of sensitivity determinations. These drugs shortened the clinical course somewhat over the control cases. Leucocytosis and antibody formation were lowered. Undesirable side effects were more common in the patients treated with aureomycin and terramycin.

Dintenfass[13] reviews in an orderly fashion the basic concepts of etiology and treatment of chronic pharyngeal infection and postnasal drip. He gives his ideas regarding treatment of the various syndromes, which for the most part, conform with generally accepted views. The importance of ventilation and drainage, plus allergic management is stressed. This article should be of value to basic students in otolaryngology.

Loeb[14] presents his experience with a series of 100 patients with epipharyngeal crypts. They are more common than presently thought and should be looked for in any patient complaining of sore throat, lump in the throat, headache, postnasal drip and crusts, blood in the throat and cough. These crypts appear as a rounded, smooth-bordered, clear-cut hole in the midline of the epipharynx, just above the level of the palate. Relief from symptoms should follow anesthetization of the crypt. In most of Loeb's cases, tonsillectomy had already been done in an effort to relieve symptoms. Treatment is directed toward obliteration or saucerization of the crypt.

Tyler[15] reports the case of a 16-year-old white girl who complained of a clicking and cracking noise in the head, migratory at first but eventually localized in the posterior portion of the mouth. It was accompanied by a grating noise in the top of the head. Physical and neurologic examinations yielded essentially negative results except for the palatal movement. With the mouth open a rhythmical 72-minute movement of the palate and uvula in a superior and inferior direction was found to be constantly present with audible clicking. The patient had no difficulty swallowing, no weakness and no

paralysis of facial muscles. Fluoroscopy revealed normal diaphragmatic movement.

This is considered a typical example of myoclonus due to damage in the central tegmental tract affecting the primitive gillslit respiratory muscles. The myoclonus resembles the gill movements of fishes. It does not extend into the somatic musculature and is likely to clear up spontaneously in a young individual.

Zinneman and Hall[16] report an interesting case of pharyngeal and laryngeal infection with Histoplasma capsulatum. A 42-year-old man noted the gradual onset of hoarseness and dyspnea necessitating tracheotomy. Because of severe sore throat deglutition was painful, resulting in loss of 25 pounds in weight. There was generalized swelling and reddening of the mucosa of the hard palate, tonsillar pillars, uvula and posterior pharyngeal wall. The mucosa was granular in appearance and of spongy consistency. There was no ulceration. Similar lesions were noted in the larynx. Biopsies revealed the characteristic histologic picture of histoplasmosis. Ethyl-vanillate 0.6 Gm. in capsules was given orally every three hours. In six weeks there was no evidence of histoplasmosis.

The natural reservoirs of Histoplasma capsulatum are the soil, dogs, rats, skunks, opossums and cats. Until recently, human histoplasmosis was thought to be a fatal infection. Clinical manifestations vary. Ethyl-vanillate is a comparatively new drug, having been used only for the past two years, and apparently successfully copes with stubborn infections of histoplasmosis. The drug is not without danger and should be used carefully.

A case of histoplasmosis involving one vocal cord was seen by one of the reviewers in 1947. This patient was successfully treated with promine and remains well today except that Addison's disease has developed as a complication of the histoplasmosis. We should be grateful to Zinneman and Hall for calling attention to the use of Ethyl-vanillate in infections of histoplasmosis.

Bucco-pharyngeal ulcerations of undetermined etiology form the basis of an interesting discussion by Harrison[17] These

recurrent ulcerations are not only painful but they also frequently defy all manner of treatment. The lesion may appear anywhere on the mucous membrane of the buccal cavity and pharynx, usually as a small red papule about the size of a pin head. Ulceration with a yellowish exudate forms rapidly and pain develops with equal rapidity. These ulcerations may be single or multiple and occasionally are fairly large. Harrison was able to obtain no information as to the etiologic factor from all laboratory tests including scrapings and biopsies. Most of Harrison's patients gave a history of severe emotional upset preceding the onset of the lesions. Numerous therapeutic agents were employed, but best results were obtained from local application of a paste made from a 0.25 mg. capsule of aureomycin in glycerine. Pain was relieved in 24 hours. The use of small doses of Roentgen-ray was of questionable value. Fortunately, such cases are relatively rare. Failure to establish the etiology of the ulceration, particularly when large, is at times disconcerting. In severe cases, or in extensive ulcerations defying all methods of therapy we have successfully used cobra venom, when available, or smallpox vaccine.

Meyrick[18] reports the case of a patient with symptoms typical of the syndrome of the sinus of Morgagni, the physical signs of which were originally described by Trotter in 1911. The patient had lost hearing in the left ear for the past two-and-one-half years and complained of vague nasal symptoms for a period of time. In the past two years he had had pain on the left side of the face, maxilla and ramus of the left mandible, and for four months, diploplia and difficulty in moving the jaw. A tumor was present in the nasopharynx and a biopsy taken through the nasal passage established a diagnosis of malignant endothelioma. Deep Roentgen-ray therapy was instituted, and 12 months later all signs and symptoms had subsided except a slight degree of deafness in the left ear.

Delaney and Nelson[19] consider the anginose, or pharyngeal type, to be the most common of the many cases of infectious mononucleosis seen. Infectious mononucleosis is frequently confused with acute tonsillitis, peritonsillar abscess, acute pharyngitis, Vincent's infection, or even diphtheria. The anginose type of infectious mononucleosis, according to Delaney

and Nelson, is characterized by diffuse pharyngitis and tonsillitis.

Prostration is no greater than in simple tonsillitis or pharyngitis, and severe respiratory difficulty is uncommon. Delaney and Nelson report a case of infectious mononucleosis which required tracheotomy. Decannulation was possible on the fourth day because of good response to terramycin, cortisone and gamma globulin. Cortisone was given for the possible effect on edematous pharyngeal tissue, but no reason is given for the administration of gamma globulin.

Wolf[20] correctly observes that prior to the advent of antibiotics, pharyngitis was treated primarily by warm gargles or application of a solution to the posterior pharyngeal wall. Instead of eliminating these methods the antibiotics have complicated the treatment of this infection. Troches are now available containing almost any desired antibiotic or groups of antibiotics with a local anesthetic. Since sensitization frequently arises from prolonged frequent contact with an antibiotic, the local use of any systemically employed antibiotic is contraindicated. Reaction to the "caine" group of anesthetic agents is not uncommon. Such reactions may take many forms, and the symptoms of pharyngitis may persist as a result of the use of these drugs. Wolf concludes that since parenteral medications are so often used successfully in infections, it is probably best to avoid intraoral use of any possible sensitizing agent, whenever possible.

In a preliminary survey of persistent sequelae of bulbar poliomyelitis an unexpectedly high incidence of persistent or recurrent disability of the pharynx was noted by Bosma.[21] In an excellent presentation he further states that two patterns of partial paralysis and associated disability of function have been observed. The more common is that of weakness of the levator veli palatini muscle with associated impairment of the palatopharyngeal sphincter. This results in a nasal quality of speech and possibly in nasal regurgitation of fluids. The less common but more critical paralysis is that of the pharyngeal constrictor musculature associated with inability to swallow. These problems are further discussed in a highly

interesting and scientific manner. The material presented by Bosma is too lengthy to review here, but the article should be read by all interested in the subject, as Bosma has devoted much time, thought, and study to this important problem.

Hansel[22] presents five cases of patients with somewhat vague symptoms referred to the arterial region of the pharynx and larynx as convincing evidence that these phenomena are vascular in nature. The clinical picture resembles that of migraine, tension and histaminic headaches. Hansel believes that the response to the administration of histamine and the symptomatic relief from the use of ergot preparations indicate that in certain patients pharyngeal and laryngeal symptoms may be of vascular origin.

Wallach and associates[23] describe an extremely contagious, previously unreported disease of children. The clinical course follows a typical pattern. The incubation period is 24 to 72 hours. During the first 36 hours the only clinical finding is a scattering of small vesicles or petechiae on the hard and soft palate and frequently on the pharynx. Following this, however, high fever, cervical lymphadenopathy and grippal symptoms rapidly develop. During this stage, coalescence of the smaller petechiae may produce large hemorrhagic blotches. The temperature usually terminates by crisis in four or five days, the mouth remaining sore for three or four more days. Penicillin therapy has no effect whatsoever. Terramycin, if administered prior to the onset of fever, seems to reduce the severity of the ultimate clinical course; however, after the onset of fever, it apparently has no effect. No laboratory studies were made during this epidemic. Hospitalization was not required; there were no fatalities and no serious complications.

Recent studies have indicated interference with the action of penicillin or streptococci by chloramphenicol. Walker[24] was unable to demonstrate this interference in a controlled series of 71 patients with acute streptococcal pharyngitis. The series was divided into three groups: one received penicillin in usual doses, and chloramphenicol in maximum doses; the second, penicillin, and chloramphenicol in minimal doses; the third, penicillin alone.

Waldo[25] states that the most common cause of acute pharyngitis is non-bacterial, the so-called "viral" pharyngitis, and requires only symptomatic treatment. Diphtheritic sore throat, which is still encountered in Utah, is preventable by immunization; early diagnosis and administration of antitoxin are imperative. Acute streptococcal sore throat presents a beefy red, edematous pharynx and responds readily to penicillin. The acute pharyngitis seen in infectious mononucleosis is commonly undiagnosed. A high index of suspicion and a positive heterophil agglutination reaction aid in diagnosis. Vincent's pharyngitis presents dirty, punched out, ragged ulceration and responds to administration of penicillin. Secondary syphilis may produce acute pharyngitis. Diagnosis in such cases is important, because insufficient penicillin therapy is harmful to the course of the disease.

Kinghorn and associates,[26] an internist, a laryngologist and a bacteriologist, report good results in 16 patients with chronic sinus disease and seven patients with "nasal catarrh" treated with autogenous vaccine and filtrate, both intradermally and locally. This study apparently took place over a period of many years.

Asherson[27] describes a characteristic syndrome manifested by a characteristic pharyngogram. This syndrome is produced by incoordination or failure of relaxation of the cricopharyngeus muscle (achalasia) during the act of swallowing. Any lesion that interrupts the persistaltic wave preceding a swallowed bolus will produce this syndrome. Paralysis of the pharyngeal plexus following poliomyelitis, paralysis of one or both recurrent laryngeal nerves, and pharyngotomy are common causes. There may be no symptoms, or symptoms may be so severe that gastrostomy is required. This is strictly a roentgenologic diagnosis. Differential diagnosis includes postcricoid carcinoma and pharyngoesophageal diverticulum. "Globus hystericus" is not mentioned. Numerous case histories and Roentgenograms are included.

According to Cockburn,[28] during August and September, 1951, 205 cases of acute nonpurulent conjunctivitis, vesicular pharyngitis, muscular ache and fever appeared in Greeley,

Colorado. Cockburn named this syndrome "Greeley's disease." The condition affected children and teen-agers and seemed to be transmitted by contact or by association with a swimming pool. No etiologic agent was found. Similar epidemics occurred during the same months in Missouri and other areas of Colorado. Many of these patients were suspected of having poliomyelitis, but the spinal fluid examinations yielded normal results. There were no complications of the disease.

Kinawi[29] reports two interesting cases of primary atypical pneumonia in sisters in whom a peculiar painless, afebrile, sterile membranous lesion of the tonsils developed ten days after resolution of the pneumonia. Both cases were thoroughly studied etiologically and clinically, and no causative agent could be found. Kinawi suggests the term "primary atypical pulmonary tonsillar syndrome" for this condition.

BENIGN TUMORS.

Davis[30] reports a case of benign pharyngeal polyp in a 19-year-old girl which caused her to complain of a feeling of "something in her throat," gagging, vomiting and loss of weight of two years' duration. Examination revealed a large, white, glistening mass extending behind the tongue into the hypopharynx. This was removed by a snare. It measured 7.5 x 3 x 1.5 cm. and was attached by a narrow pedicle to the left lateral wall of the nasopharynx. There has been no recurrence.

Guggenheim[31] reports a case of schwannoma of the pharynx, which is considered a rare disease. He was able to find only 34 similar cases reported in the domestic and foreign literature and these, which are tabulated, form part of this excellent paper. In the case reported the tumor extended from above the soft palate to just below the level of the epiglottis and after preliminary tracheotomy a submandibular incision on the left was made back to the angle of the jaw curving gently around to continue downward along the anterior border of the sternocleidomastoid muscle. The external carotid was ligated and dissection continued until the tumor was exposed and removed. Upon intraoral examination later, Guggenheim discovered some of the tumor still present, and this was re-

moved perorally. The origin of the tumor is discussed, and Guggenheim advocates a combined approach in all cases of schwannoma of the pharynx.

According to Tingwald[32] only 20 cases of polyps arising in the oropharynx and hypopharynx have been recorded in the literature. He describes an additional case of a 48-year-old white man with a one year history of a lump in the left side of his throat which caused him to have a constant desire to clear his throat. The lump was caused by a polyp whose pedicle was attached to the left lateral pharyngeal wall at the level of the arytenoid cartilage. After fixation the specimen measured 4.5 cm. in length and 1.5 cm. in diameter. Previous pathologic diagnoses of these lesions include lipoma, myxoma, fibroma, fibro-epithelial polyp and benign fibromatous polyp. In two of the reported cases the lesions were multiple. Symptoms reported include regurgitation of the polyps, intermittent dysphagia, lump in the throat, throat clearing, cough, dyspnea, hoarseness, wheezing, nausea and vomiting. Three patients died of asphyxia due to aspiration of these polyps and it is primarily for this reason that they achieve importance.

Farrar[33] classifies fibromas of the nasopharynx into two distinct types: the first is the simple fibroma, which may arise in the nasopharynx or grow into it from the nose. It has a variable histologic picture, is vascular, may occur at any age and in either sex. The second type is the juvenile basal fibroma of the nasopharynx. It arises only in the nasopharynx and occurs nearly always in adolescent boys. The site of attachment is usually on the sphenoid. These tumors are very vascular, the blood vessels lacking muscular wall in parts. Occasional surgical removal may be accompanied by uncontrollable hemorrhage.

Farrar reports a case of simple fibroma of the nasopharynx occurring in a boy aged 13 years. The tumor, which was smooth and firm to touch, filled the nasopharynx and was attached to the posterior tip of the right inferior turbinate. It was readily removed by snare and a brisk hemorrhage was controlled with packing. Such tumors are comparatively rare,

only six being found in a review of 211 tumors of the nose and nasopharynx.

In an interesting article on benign neoplasms of the nose, Handousa[34] presents the main features in 73 consecutive cases seen in private hospitals. The tumors encountered were osteomas, hemangiomas, fibromas, papillomas, chondromas, lymphomas and myxomas. Each of these groups of cases is described accurately, and the operative procedure and results are tabulated. Comments pertinent to the type of tumor described are offered.

A new surgical approach to fibroma of the nasopharynx is described and illustrated by Kremen;[35] however, before describing his operative approach, he thoroughly discusses angiofibromas or the so-called juvenile fibromas of the nasopharynx and correctly states that they occur more frequently in young pubescent males. The usual symptoms produced by these tumors are nasal obstruction, changes in voice and hearing, and frequent bleeding from the nose or nasopharynx. The lesion grows rapidly during adolescence, but after maturity the prominent vascular channels tend to become smaller, and the tumor regresses considerably. Although the lesion is not malignant histologically, its growth and expansion are at times responsible for changes in facial contour. It is well known that these tumors usually arise from the posterior superior wall of the nasopharynx. Because they do not respond satisfactorily to radiation and continue bleeding excessively, it frequently becomes necessary to remove them surgically. Because Kremen was critical of operations heretofore used for removal of nasopharyngeal fibromas, he devised a technique which is described and illustrated. He makes a vertical incision in front of the ear and down the neck just anterior to the sternomastoid muscle. Another incision beneath the angle of the mandible is of assistance in retracting flaps and exposure. The external carotid is ligated. The masseter muscle is exposed and incised to the periosteum, which is elevated, and the mandible is then transected with a Gigli saw about 1 cm. below the notch formed by the coronoid and condyloid processes. The edges of the cut mandible and the pterygoid muscle fibers are separated. This exposes the tubulomuscular wall

of the nasopharynx, which is incised longitudinally on its lateral wall. The tumor is thus exposed and extirpated. The mandible is brought together with stainless steel wire. Two cases in which this operation was successfully employed are reported.

Much credit is due Kremen for the development of a new technique which he claims is advantageous; however, the technique appears complicated and long in contrast to the time honored methods which have served excellently in the past. We have had opportunity to see many juvenile nasopharyngeal fibromas, and all presented problems. Controlling hemorrhage has been the most important, and when accomplished, we have found that repeated electrocoagulation and repeated implantation of radium needles or radon seeds into the tumor have eventually reduced the vascularity and size of the tumor so that it could be successfully removed by snare.

An unusual case of transitional cell papilloma occurring on the nasal and oral portions of the post-pharyngeal wall is reported by Radcliff.[36] This tumor, which extended from the basisphenoid to about one-inch above the epiglottis, appeared as a papilliferous mass attached to the posterior pharyngeal wall. Through a transverse palatine incision the growth was removed by diathermy excision without difficulty or complications. The pathologic report was transitional cell papilloma with no evidence of malignancy. Radcliff reviews the literature on similar cases showing that there is some definite difference of opinion on the histologic aspects of this type of neoplasm.

Manning[37] reports the case of a 25-year-old man with a solitary neurofibroma bulging into the pharynx and neck associated with Horner's syndrome. The mass was removed, and there was no evidence of recurrence eight months later. Five other cases collected from the literature are reviewed.

MALIGNANT TUMORS.

Dysphagia associated with hypochromic anemia, which has been called Plummer-Vinson syndrome, Patterson-Brown-Kelly syndrome, and sideropenic dysphagia, is common in Sweden.

Lindvall[38] gives an excellent discussion of the etiology, pathology and typical roentgenographic observations in such patients. In 300 cases of hypopharyngeal or upper esophageal carcinoma observed in women, over 90 per cent gave a history of dysphagia and hypochromic anemia. Valve-like obstructions, webs and actual strictures are demonstrated in the hypopharynx and upper esophagus roentgenologically in many patients. When a carcinoma occurs, it is practically always proximal to these changes. Eight cases are presented in which roentgenologic changes of siderøpenic dysphagia were demonstrated many years prior to the development of the carcinoma.

In a review of the literature Holinger and Rabbett[39–41] found nine cases of malignancy of the pharynx and larynx in which the tumor developed in an area previously irradiated for a benign process and present three cases of their own. The latent period appears to be 20-35 years. Cases of carcinoma of the skin developing in previously irradiated areas are well known. Recently, reports of bone sarcomas in areas treated three to 12 years previously for tuberculous arthritis and carcinoma of the cervix developing ten or more years after irradiation therapy for a benign lesion of the cervix have appeared in the literature. Since irradiation therapy was more common for benign conditions of the neck several decades ago, more of these cases may be seen in the immediate future. They believe the sex distribution of the 12 cases may be significant (nine females and three males), since the pyriform sinus and the laryngeal lesions are seen most commonly in males and only the postcricoid lesions most frequently in females.

Vincent[42] reports the case of a patient aged six years, who died of a rapidly growing tumor of the nasopharynx two months following tonsillectomy. An extensive neuroblastoma arising in the nasopharynx, filling the upper air and food passages, and extending into the cranial cavity at several points was found at autopsy.

Rubenfeld and Winston[43] state that obtaining cytologic cells by suction has found its greatest application in gynecologic tumors. The simplicity of the procedure prompted them to try

the same technique in tumors of the nasopharynx. With the patient in the sitting position the nasal cavity is cocainized and a metal cannula is inserted along the floor of the nose until it strikes or comes in contact with mucosa or tumor. Suction is produced by a 55 cc. syringe as the cannula is moved around, and the trauma produced by the pressure and motion of the cannula is sufficient to obtain a few fragments of tissue. The simplicity of the technique recommends its use.

In an excellent article Hultberg[44] describes Radiumhemmet's method of treatment in hypopharyngeal cancer. He points out the importance of first distinguishing between cancer of the upper and lower portions of the hypopharynx, the line of division being at the level of the upper border of the cricoid cartilage. The upper group comprises carcinoma of the edge and anterior surface of the epiglottis, the aryepiglottic fold, the arytenoid region and the pyriform fossa. To the lower group are assigned postcricoid carcinomas and tumors occurring at the entrance of the esophagus. Since 1930 fractionated but not protracted Roentgen-ray treatment has been used at Radiumhemmet. The technique is described in detail, one field being radiated each day, and it is estimated that the total dose required to produce complete regression of the cancerous growth is from 5700 to 6000 R. administered over a period of 28 to 32 days. Between 1939 and 1947, 322 (119 males, 203 females) patients with hypopharyngeal cancer were treated in this way. The incidence of five-year cures in the entire series was between nine and 10 per cent; however, in the 65 patients treated in 1948 and 1949, the five-year cure rate increased to 14.5 per cent. This indicates that the results are improving. Carcinomas in the lower part of the hypopharynx are usually regarded as incurable. Hultberg concludes that if it were possible to persuade people to desist from smoking and drinking strong spirits, fewer cases of pharyngeal carcinomas would be seen.

Welin[45,46] informs us that in Sweden the hypopharynx is a relatively common site for cancer. Since 1940, 510 such cases were seen at the Karolinska Hospital. Their records show that the malignancy in men is more often localized in the upper portion of the hypopharynx whereas in women the opposite is true. This is significant and probably connected with the rela-

tively high incidence of the Plummer-Vinson syndrome in women, which is considered by Welin to be a precancerous condition. Tumors localized in the postcricoid portion of the hypopharynx are best diagnosed and their limits defined by radiologic examination. Since radiologic techniques differ in the various countries, Welin describes the technique used in Sweden. This is illustrated with diagrams and radiologic views. The diagnosis of carcinoma of the hypopharynx is made radiologically, and the extent of the tumor and the degree of invasion into the normal surrounding tissues is indicated to the clinician. Patients with Plummer-Vinson syndrome are requested to return every six months for follow-up examinations. This has resulted in the diagnosis of carcinoma before any new symptoms are produced. Roentgen-ray therapy is discussed, and a warning regarding late reactions following irradiation is sounded.

Duncan[47] reports a case of mixed tumor of the hypopharynx treated by local excision. Local recurrence, spread and pulmonary metastasis developed subsequently, and were treated palliatively with Roentgen-ray therapy.

Heatly[48] reports two cases of plasma cell tumors involving the maxillary sinus, both of which were remarkably vascular.

Plasma cell tumors involving the upper air passages are rare and are commonly associated with multiple myeloma. They may occur as localized submucosal tumefactions or as pedunculated polyps. They are quite unpredictable in behavior, and neither gross nor microscopic appearance is of value in predicting the degree of malignancy. The consensus is that they are best treated by Roentgen-ray therapy and local excision when this does not involve a radical surgical procedure. They should be considered malignant. Local and distant recurrences are possible many years after apparent cure of the initial lesion. All patients with plasma cell tumor should have a skeletal survey, marrow studies and a search for Bence-Jones protein to differentiate it from multiple myeloma.

Negus[49] believes that for carcinoma at or near the mouth of the esophagus, involving the posterior surface of the larynx,

radical removal of a segment of the pharynx and upper end of the esophagus, together with the whole of the larynx is the only adequate surgical measure.

The primary principles involved include removal of the growth in one block as soon as possible. No preliminary measures to reconstruction are taken before the carcinoma has been eradicated. The entire procedure including the reconstruction is done in one stage.

The *primary* reconstruction is accomplished by filling the food passage defect with a plastic tube, tapered from above downwards, covered with a free split-skin graft. This plastic stent is allowed to remain for two months.

Complications include infection, fistula and stenosis. Biotherapy is largely capable of preventing infection. Stenosis may be benefited by bouginage but, preferably, the lumen should be re-established by formation of flaps.

Of 13 patients seen since 1949, only seven were considered potentially curable. Of these, two died one year after operation from local recurrence, and the other five are well three and one-half, two and one-half, and two and one-quarter years after operation.

Moore and Faulkner[50] call attention to the fact that extrinsic carcinoma of the larynx is amenable to pharyngolaryngectomy in only a limited number of cases and then rarely with a permanently successful result. Patients should be selected carefully with the idea of accomplishing both surgical extirpation of the lesion and plastic repair of the esophagus and pharynx in the shortest possible time.

The advantages and disadvantages of the Negus method of single-stage repair are discussed. It appears that the disadvantages could be overcome merely by improving existing techniques. In patients who survive the disease and the operation, stricture of the newly formed food passage may become a problem whose solution lies in several choices of surgical techniques. Four such methods are discussed in some detail. Moore and Faulkner describe their method of two-stage reconstruction in which the skin flaps to be used in the repair

are sutured to the esophagus before it is cut across. In this way the cut end of the esophagus is "fixed" and does not retract, making anastomosis as low as the level of the aortic arch possible. The second stage is done as soon as the wound has healed and the sutures are removed. This method of reconstruction is particularly suitable in those patients whose tracheal defect is a major problem.

Irradiated skin should never be used in the repair. Fistulas, if small, will usually close spontaneously if the passage of food is diverted via a Levine tube. Larger fistulas may be closed by turning in local flaps. It is important to look for the development of hematomas beneath the skin flaps postoperatively. All saliva should be ejected into a sputum bowl and not swallowed.

Anesthetic problems and difficulties associated with this procedure are discussed.

Cade,[51] who has treated 403 cases of carcinoma of the pharynx in the past 22 years, believes that Roentgen-ray therapy offers little in its treatment. He considers radical surgical excision including the larynx, upper esophagus and pharynx the treatment of choice in operable cases. Raven discusses his technique of radical two-stage operation used in 24 patients. Three methods of closure of the pharyngoesophageal defect are available: partial closure with the original skin flap, delayed closure by tube pedicle grafts, or immediate closure by the use of a skin graft over a plastic tube. The latter is the ideal.

Patients with advanced cancer of the oropharynx, hypopharynx, larynx and cervical esophagus have a miserable terminal illness and death. Palliative surgical procedures with or without irradiation rarely improve their lot. Braund and Cocke[52] believe that in selected cases extensive surgical excision, with *en bloc* removal of the larynx, upper esophagus, portions of the oropharynx and hypopharynx as well as unilateral or bilateral radical neck dissections, not only provides satisfactory palliation but also offers hope for cure. Reconstruction of the upper esophagus, re-establishing pharyngoesophageal continuity, is accomplished by the use of parallel

tube grafts raised from the anterior thoracic wall. Details of technique, and preoperative and postoperative care are given.

Gooddy[53] emphasizes the importance of careful inspection of the nasopharynx in cases of obscure facial pain. The initial symptoms of nasopharyngeal cancer are neurologic in 16 per cent of cases, Vth Nerve symptoms being by far the most common. Cawthorne[54] reviews the diagnostic procedure employed in tumors of this area. Professor McWhirter[55] stated that cancer of the nasopharynx is a rare lesion and not frequently seen by the medical students or by general practitioners.

The pharyngeal grooves are the terminal portions of the lateral food channels for directing food past the larynx into the esophagus. In an interesting article on cancer of this area, Lederman[56] points out the difficulties of trying to define the pharyngolaryngeal groove and particularly the sinus pyriformis for purposes of classifying tumors in this region. He concluded that the acceptable anatomic boundaries for the pharyngolaryngeal groove extend from the pharyngoepiglottic fold to the esophageal opening at the level of the lower border of the cricoid cartilage. He further attempted to distinguish tumors arising in the pharyngolaryngeal groove or "pyriform fossa" into an upper group and a "worse" or lower group. Signs and symptoms occurring in each group are briefly discussed as well as the radiologic appearances. During the years 1933-1951, 318 laryngopharyngeal tumors were seen and of these 119 or 37 per cent arose from the sinus pyriformis. Lederman states that teleradium has been the treatment of choice, only three patients in his series having undergone laryngopharyngectomy after radiation. The incidence of three-year survivals was surprisingly good, but it diminished considerably for survival from five years, owing to recurrences or metastasis. Only 6.5 per cent survived five years. In review, it must be remembered that cancer of the pyriform fossa, when first seen by a laryngologist is as a rule extensive, as the signs and symptoms are such that early diagnosis is very unlikely. Many of the patients treated by radiation have such extensive lesions that they are inoperable and, therefore, any survivals represent good salvage.

According to Conley[57] a one-stage operation for the treatment of patients with extensive cancer of the pharynx, hypopharynx, postcricoid areas and cervical esophagus has been developed. It consists in immediate re-establishment of continuity between the fenestrae in the oral pharynx and cervical esophagus by anastomosing a free, thick-split, tubed skin graft to these structures.

Indications for this procedure are extensive cancer of the cervical esophagus, of the pharynx and hypopharynx, and of the extrinsic larynx and pharynx. Plastic reconstruction is indicated whenever the cancerous process occupies more than 70 per cent of the circumference of the pharyngeal food passage. In esophageal lesions it is rarely possible to do less than total circumferential excision. The operation is not warranted in areas that have been heavily irradiated or are grossly secondarily infected.

Important steps in the technique include careful end-to-end anastomosis of the free skin tube to the circumference of the pharyngeal and esophageal mucosa. The longitudinal suture line of the tube is approximated to the prevertebral fascia of the unoperated side of the neck. This insures maximum support and nourishment to the tube which is also enhanced by the covering inferiorly with the lobes of the thyroid.

Complications include infection, fistula and stenosis. The latter is the most serious and may be prevented largely by use of plastic or wire mesh stents as an onlay for the tubed skin graft.

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THE MANAGEMENT OF EPISTAXIS.

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Epistaxis is not a new and interesting condition, and there is nothing startlingly new and interesting to report about its management. It is such a common condition, and fortunately, in the usual instance, so simple to deal with, that we sometimes tend to forget that occasionally bleeding from the nose is of serious import and may require the best medical and surgical skill and acumen for solution of the problem it presents. In rare cases, when the hemorrhage is profuse or prolonged, epistaxis may constitute a difficult surgical emergency.

For this reason, it seems well, in the case of this and other common conditions, to review from time to time our own and others' experiences and to bring together what has been learned about the most effective methods of management. On looking back over the years during which I have been engaged in the practice of otolaryngology, there has been no outstanding innovation which marked a complete change in the methods of handling nasal bleeding, but there have been numerous modifications and improvements, as the result of various medical and surgical developments.

Before discussing these measures in more detail, it probably is well to enumerate the principal causes of hemorrhage from the nose. The local causes include: *1.* trauma, either accidental or surgical, or from picking, rubbing, and blowing the nose; *2.* acute and chronic infections; *3.* perforation of the septum with repeated hemorrhages from the denuded area; *4.* a spur or deflection of the nasal septum; *5.* neoplasms in the nose or nasopharynx, and *6.* hemorrhagic telangiectasis.

The general or systemic causes for bleeding from the nose are: *1.* hypertension and arterial changes, such as arterio-

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sclerosis; *2.* increased venous tension, as in emphysema, whooping cough, bronchitis, tumors of the neck or chest, aneurysms of the neck or chest; *3.* blood diseases, including leukemia, hemophilia, pseudo-hemophilia, multiple myeloma, pernicious anemia, secondary anemia, purpura, etc.; *4.* cardiac diseases, particularly mitral stenosis and rheumatic fever in children; *5.* a lowered prothrombin time due to hepatic disease or to the use of dicumarol or other anticoagulant drugs, acetyl salicylic acid, and the like; *6.* acute infectious diseases, especially at their onset; *7.* menstrual disturbances resulting in vicarious bleeding; *8.* scurvy and other types of avitaminosis, and *9.* high altitudes.

A relatively common site of posterior nasal bleeding in older persons is an area of dilated veins underneath the posterior end of the inferior turbinate. Woodruff has designated this area as the "naso-pharyngeal plexus." Bleeding from the lateral nasal wall is usually from the lateral branch of the sphenopalatine artery. The medial branch of this artery, sometimes called the nasopalatine artery, may also be the source of bleeding from the anterior inferior portion of the septum. Other types of nasal bleeding include that from behind septal spurs and deflections, diffuse bleeding from the nasal mucous membranes, usually seen in patients with blood dyscrasias, and bleeding from the anterior ethmoidal artery or vein, which is most frequently caused by trauma.

The incidence of epistaxis is highest in children but seldom is serious in these young individuals. The more severe forms of epistaxis are seen in older persons and are often related to the presence of degenerative vascular and other diseases. In a series of 212 cases of severe epistaxis requiring hospitalization, reported by Hallberg from the Mayo Clinic, the incidence increased directly with age. More than 40 per cent were over 60 years of age, and of this group, 65 per cent were from hypertension.

The multiplicity of causes for nasal bleeding and the various sites of hemorrhage make it obvious that the management of each case must be individualized. The severity ranges from a slight dripping of blood to profuse, shock-producing, and even fatal hemorrhage, and treatment must be applied accord-

ingly. Whatever the cause or source of the hemorrhage, the aim of treatment is to stop the bleeding as promptly as possible. If the cause is purely local, the difficulty of the problem depends upon the site and type of vessel involved and the amount of blood lost. When the bleeding is secondary to systemic causes, thorough clinical studies and the collaboration of otolaryngologist and internist may be necessary before the problem is satisfactorily solved.

Therapeutic Armamentarium.—In the clinical management of epistaxis, a variety of coagulants are available for use according to the indications in the individual case. A variety of substances are used for nasal packing, for some of which special techniques have been recommended.[7,14,15,19,57] Besides these, there are the agents for chemical cautery and electrocautery, by the actual cautery or by electric coagulation. When medical measures and cautery do not suffice to control the bleeding, then ligation of the blood vessels is indicated. The surgical procedure most frequently employed is ligation of the external carotid artery; in rare instances, interruption of the external maxillary or one of the ethmoid arteries may be necessary, depending upon the site of the hemorrhage.[5,21,23,25,32,42,46,47,49,53,55,56,57]

Among the coagulants which have been recommended for use in the treatment of epistaxis are vitamin K,[51] which is necessary for the synthesis of prothrombin; protamine sulfate (Lilly), which is an antidote for heparin; vitamin C; Cortisone,[1,9,11,34,39,48] which has been shown to affect the coagulation of the blood and to exert a favorable effect in patients with pseudohemophilia, or prolonged prothrombin time; Koagamin (Chatham), which is an aqueous solution of oxalic and malonic acids, with 0.25 per cent phenol;[12] Neo-hemoplastin (Parke-Davis); snake venom, useful in capillary bleeding by direct effect on clotting of fibrinogen; oxalic acid used intramuscularly or intravenously,[36] and pituitrin.[27]

The substances used for nasal packing, besides surgical gauze, include oxidized cellulose (Oxycel);[16,17,35,43] gel foam;[45] thrombin,[13,54] alone or in combination with oxidized cellulose or gel foam; Simpson splints; rubber balloons filled with air,

water or vaseline gauze; sponge;[15] cotton, and salt pork (for the hypertensive patient).[10,38]

Substances used for chemical cautery are silver nitrate, trichloracetic acid, neutralized chromic acid, basic ferric sulfate or subsulfate (Monsel's solution), compound tincture of benzoin and burnt alum.[12,62]

Management of Epistaxis in Children.—It has often been noted that simple nose bleed will stop spontaneously, regardless of treatment used, or even without treatment. Although this is true, it must also be remembered that to the patient this fact may not be known or obvious. Whenever the bleeding from the nose is of sufficient severity that a physician is consulted, it is to the patient, or to his family, a serious matter and sometimes a frightening occurrence. For this reason, reassurance is the first step in treatment. If the patient is at home, instruction should be given that he should be placed in the proper position, that is, that he should sit up and bend forward. If he lies on his back, the stomach may fill with blood, causing faintness and nausea. In cases of simple nosebleed from the anterior septum, blowing out the clots and pressing the anterior nares together for a few minutes may be sufficient to control the condition.

Whenever intranasal manipulation or packing is needed, the administration of a sedative is indicated, whether the patient be a child or an adult. Once his anxiety is relieved and he becomes relaxed, whatever measures are necessary to control the bleeding are much more easily accomplished. In cases of anterior nasal bleeding from Kiesselbach's or Little's area, the bleeding vessel can frequently be seen, if the hemorrhage is not too profuse; then the area is cocainized and cauterized either with one of the chemical agents or the electric current. In attempting to control hemorrhage from the anterior part of the nose of a child, my associates and I prefer the use of chemical cautery to electrocoagulation. In many instances, we use a solution of 50 per cent silver nitrate which is immediately neutralized by sodium chloride solution and covered with holocaine and adrenalin ointment. Some authors suggest the use of a silver nitrate stick or chromic acid or trichloracetic acid;[2] the latter two should be neutralized by a solution of sodium bicarbonate.

When the hemorrhage in the anterior portion of the nose is so severe that it is impossible to find the bleeding point, the nose is packed with cotton pledgets saturated with cocaine and adrenalin until the hemorrhage ceases. Then an attempt is made to find the bleeding point in order that the area may be cauterized.

When the hemorrhage is from the anterior superior part of the nose, and especially when there is a history of recurrent or profuse bleeding, we have found it most satisfactory to use "spot" packing of oxidized cellulose or gel foam saturated with thrombin and held in place with a cotton sponge and a small piece of adhesive placed over the nostril to prevent the pack from being dislodged by sneezing. The cotton should be saturated with penicillin solution to reduce the chance of infection. We also administer an antibiotic systemically if the pack is to be left in place as long as 24 hours.

In case of recurrent nasal bleeding in children, which the parents usually say is spontaneous and frequently comes on at night, there is often a discharge with crusting of the septum near the anterior nares which may be due to infection or to allergy. The child with allergic rhinitis has a tendency to rub the nose to relieve the itching sensation and to pick at the crusts with the finger. Excessive drying of the nasal mucus greatly reduces its protective value and renders the capillary bed more accessible to trauma. A measure devised by Hilding and recommended by Hunnicutt[37] is helpful in preventing crusting and allowing the tissues to return to normal; this is to place a light plug of dry cotton in the nostril for a few minutes several times a day. In cases in which any nasal discharge is present, it is important that appropriate treatment for the underlying condition be instituted.

An anterior deviation of the nasal septum is frequently a contributory cause in recurrent epistaxis, since it increases the likelihood of trauma of the convex side, and often there is also an accumulation of mucus with crust formation on the side of the convexity.

In all instances of continued or recurrent epistaxis in children, a thorough clinical examination and appropriate lab-

oratory studies are indicated, to determine whether the bleeding is being caused by some systemic disease. Nasal hemorrhage of systemic origin in children is frequently noted in diseases of the blood, liver or kidneys, and in acute infectious diseases, particularly in acute rheumatic fever. In such instances, treatment of the underlying disease is of paramount importance, with the local measures which are necessary for the control of bleeding from the nose.

Kugelmass[44] reported that nontraumatic, nonirritant nosebleed occurred in about one-third of rheumatic children observed over a 15-year period, especially at the onset of acute episodes. This hemorrhage is attributed to increased vascular fragility of the nasal mucous membranes, which is said to be pronounced during puberty and prevalent during active carditis. He reported favorable effects in such cases from the use of vitamin P (rutin) derived from lemon rind and administered as tablets of eriodictyol or capsules of the chalcone. Kugelmass said that the medication increased capillary resistance above the critical level for bleeding but not to the normal level, and was effective in diminishing the frequency and severity of the rheumatic epistaxis.

Since Cortisone has been reported to have a favorable effect during the acute attack of rheumatic fever[34] which is the time that epistaxis is usually most pronounced, and since it also apparently increases the coagulability of the blood,[11] it is to be expected that its administration during the acute phases of rheumatic fever may have an influence in controlling the epistaxis observed in this condition.

When the history, general physical examination and laboratory findings indicate that the nasal bleeding is not due to any systemic disease, we believe it useful to prescribe large daily doses of vitamin K and of vitamin C and to increase the quantity of fruit juices, in the diet, even though there is no evidence of clinical nutritional deficiency, and the diet is thought to be adequate.[27,81]

Treatment of Epistaxis in Adults.—The type of nasal hemorrhage seen in children may also be seen in mature individuals, although with considerably less frequency. The milder

forms of epistaxis in adults are treated as they are in children. The more severe episodes of hemorrhage from the nose are encountered, as a rule, in elderly individuals with hypertension and other degenerative diseases. The bleeding in such cases is often from the upper or posterior areas, where it is difficult to find the eroded vessel.

Quite frequently, patients with severe epistaxis have been bleeding profusely for several hours, and sometimes for several days, before they are seen by the otolaryngologist. For this reason, it often is necessary to administer tranfusions or other countermeasures against shock before the local situation can be dealt with adequately. It must be remembered, too, that sometimes the patient who presents a shock-like appearance is merely nauseated from swallowing too much blood. If there is any uncertainty as to the patient's general condition, however, transfusion of 500 ml. (average) of blood should be given.

When the bleeding is still active, any packing that has been placed in the nose is removed and the nose is cleansed with a suction apparatus under direct vision in attempting to find the source of the hemorrhage. For this purpose, I have found the Anthony-Fisher suction-cautery a very useful instrument. Cotton pledgets soaked in adrenalin and cocaine are used to shrink the mucous membrane and provide anesthesia for manipulation.

On some occasions in my experience, when using the suction apparatus in cases of this type, the nasal hemorrhage encountered was so profuse that it was impossible to remove the blood quickly enough to determine the area from which it originated. In such instances, insertion of a post-nasal pack, followed by firm packing of the nose, was the only procedure that could be carried out.

One-inch gauze, carefully and firmly applied in layers, is the material most often used for packing the anterior portion of the nose. Hallberg[27] suggests that the gauze be heavily covered with petrolatum. Others have recommended soaking the packing material in peroxide[7] or in tannic acid and antipyrine solution.[61] In our work, we prefer to use a gauze pack-

ing with 5 per cent sulfathiazole and to cover the post-nasal pack with 5 per cent sulfathiazole ointment. Penicillin or other appropriate antibiotic is administered while the pack is in place, to prevent infection.

With the use of antibiotics and the sulfonamides, packing may be kept in the nose for a week or longer, without danger of infection. If the bleeding is completely arrested by the packing procedure, sufficient healing takes place so that the bleeding point is permanently sealed, and, when the packing is removed, the nasal mucous membrane usually appears to be in very good condition. In our experience, the instances in which gauze packing was left in the nose for a week, or thereabouts, were those in which severe bleeding had occurred for a protracted period before we saw the patient, and it was deemed advisable to restore blood volume and electrolyte balance and to administer vitamins before any attempt should be made to find the bleeding vessels.

There are numerous instances when the Foley catheter, the Anthony-Fisher post-nasal air balloon or the Cooper Rose inflation plug may be preferable to use of a post-nasal tampon and nasal packing with gauze or sponge. Numerous other packing materials have also been suggested. Dixon[15] has advocated the use of pliable marine sponges, applied piecemeal, for the control of epistaxis. Packing with salt pork[10,38] for the control of bleeding in the hypertensive patient has been widely recommended in the literature, but I have never been impressed with the value of this measure. I tried it on numerous occasions, but have long since discontinued any attempt to use it. I found that these plugs were difficult to keep in place and very often did not control the hemorrhage.

During the past few years, there has been considerable interest in the use of some of the newer hemostatic agents, such as oxidized cellulose,[16,17,35,43] fibrinfoam,[4,18,58] and gel foam[45] for packing in the case of severe nasal hemorrhage. Houser[36] recommended using a strip of oxycellulose ½-inch wide in sufficient quantity to exert the necessary pressure for control and stated that the gelatinous mass could be removed at the end of 24 hours, or that when used with a post-nasal pack,

the latter may be removed in 24 hours, while the absorbable cellulose is left *in situ.*

On the basis of my own experience, my opinion is that neither oxidized cellulose (Oxycel) nor gel foam should be used in the nose in large amounts, such as in the control of severe hemorrhage or following submucous resection. One unhappy experience resulted in a slight injury to the mucous membrance when I was attempting to remove the jelly-like mass by means of suction and forceps. The patient now has a moderately large perforation on the septum which she is unable to forget, because of the crusts that tend to form. On the other hand, when the Oxycel or gel foam may be used in small quantities for "spot" packing or in situations where the packing may be applied without completely obstructing the nasal passage, it is quite useful. A small strip of Oxycel or gel foam saturated with thrombin is useful in controlling the bleeding following the removal of nasal polyps. In this instance, however, if the packing is not loosely applied, the patient will complain of severe headache that is most difficult to control.

Thrombin[13,54] has proved itself useful for sealing the mucous membrane flaps following submucous resections. This is accomplished by spraying a small amount of the liquid thrombin between the flaps and gently pressing them together; in most instances, this procedure is sufficient to control the bleeding. It should be emphasized that thrombin is never injected, but is always used as a topical application for the control of hemorrhage from small superficial vessels. If bleeding should occur following a submucous resection, it is well to use Simpson splints or rubber finger cots packed with vaseline gauze.

In instances in which the bleeding point cannot be located and/or cannot be cauterized satisfactorily, or in those in which bleeding recurs following nasal packing, then additional local manipulation, systemic treatment, and appropriate procedures, which may include ligation of a principal artery, must be carried out, according to the site of the hemorrhage and the indications in the individual case.

A procedure that has been recommended[3,22,50] in some cases in which routine cauterization is not successful is the injection of sclerosing solutions underneath the mucosa around the bleeding point. Among the solutions used for this purpose are 0.5 milliliter of 10 per cent phenol in almond oil; Sylnasol, 0.25 to 0.5 milliliter, and 0.1 milliliter of a saturated (16.6 per cent) solution of quinine lactate.

The epistaxis due to telangiectasia (Osler's disease) presents a special problem in treatment.[20,41,60] There may be multiple bleeding points from angiomas situated in the nasal mucous membranes. Packing may aggravate the bleeding because of injury to the mucous membranes. As a temporary measure, most authorities agree that the rubber balloon is most satisfactory in controlling the hemorrhage in this type of case. The local lesions may be treated by electrocoagulation, which is preferable to the actual cautery or chemical corrosive agents. A combination of beta radiation and deep Roentgen-ray therapy has been advocated for treatment of the local lesions by Crowe and his associates at Johns Hopkins. Repeated blood transfusions frequently are necessary in this condition to combat the anemia resulting from the frequent hemorrhages.

In certain blood dyscrasias, such as acute leukemia and multiple myeloma, there may be diffuse bleeding from the mucous membrane, including the nasal mucosa. In these cases, topical application of thrombin and thromboplastin is often useful. Since packing traumatizes the membranes, this is not resorted to unless the bleeding is so severe that it is necessary in order to save life.[27]

Cortisone has been used with favorable effect in idiopathic thrombocytopenic purpura,[1,34] allergic purpura,[1] chronic hypoplastic anemia[34] and pseudohemophilia.[39] Epistaxis is a frequent manifestation in these blood dyscrasies, and has responded, along with other hemorrhagic symptoms, to the administration of cortisone or ACTH.

Kirschner[42] called special attention to the bleeding which may be caused by polyps broadly attached to the anterior portion of the septum, and outlined the following procedure:

"The base is cocainized and separated from its subjacent layer by gauze pledgets soaked in stryphnon or liquor ferri. These are left in place over the site of the hemorrhage until a conglomeration forms. Pressure is applied from the external nostril and after a few minutes, the conglomerate can be removed. Further procedures are not as a rule necessary." Kirschner also cautioned that galvanocautery and strong caustics should not be used in the poorly nourished anterior portion of the septum, since they involve danger of perforation.

Hunnicutt[38] has recommended crushing the turbinate over its entire length as an efficient method of stopping the bleeding from an artery going to the inferior turbinate. The artery is at the surface on the lateral and inferior aspect just as it enters the turbinate at its posterior end, and it is at this point that the crushing obliterates the vessel.

Septal bleeding behind spurs and deflections may require a submucous resection as preliminary treatment before the bleeding point can be found. In numerous instances, the submucous resection alone is sufficient to control the hemorrhage. For patients who have anterior septal perforations with crusting and repeated bleeding, Hallberg[27] recommends a modified submucous resection with removal of exposed cartilage around the edges as the treatment of choice. Beinfield[6] lists the theoretical reasons why submucous resection may control nasal hemorrhage as follows: *1.* Bleeding not controlled by a postnasal pack may arise from the dilated plexus of veins underneath the posterior end of the inferior turbinate[61] and may be stopped by elevation of the mucous membrane during a submucous resection; *2.* It may be that infiltration of the anesthetic raises the mucoperiosteum of the septum, sphenoid and floor of the nose and temporarily produces pressure upon the vessels, long enough to form a clot, in addition to the vasoconstrictor action of the epinephrine in the solution; *3.* Postoperative tissue reaction to the infiltration and surgery may produce edema that constricts the vessels by its pressure; *4.* Elevation of the mucous membrane from the septum may change the position of the bleeding vessel, permitting it to retract or thrombose; *5.* The elevated septal flaps may alter

the relationship of the sphenopalatine arteries to the rigid resected bony septum when the flaps heal together, giving the arterio-sclerotic vessel an added advantage of mobility to retract with whatever slight elasticity it may still have.

When the medical measures and minor surgical procedures already outlined fail to control severe nasal bleeding, then recourse must be had to ligation of one of the arteries supplying the nose, above the bleeding point. The use of ligation of a main artery for the control of epistaxis is not new, nor are the surgical procedures specific for this purpose. The incisions and techniques used are the same as those employed in operation on the sinuses or neck for other reasons, such as the eradication of infectious or malignant lesions.

Although in the earlier literature there were numerous reports of ligation of the common carotid or internal carotid arteries for nasal hemorrhage, this is a formidable and often unsuccessful procedure. The ligation which is most often indicated and performed for intractable hemorrhage from the posterior portion of the nose is that of the external carotid artery.

Although it is more than probable that most of the ligations of the external carotid artery which have been carried out for treatment of epistaxis have gone unreported there have been several reports which dealt with this subject. When McKnight[49] reviewed the literature on carotid ligation in 1926, he found 17 instances in which ligation of the external carotid artery for nasal hemorrhage had been reported. Spar and Hallberg[57] noted eight additional cases in the literature and reported a series of 11 cases, observed in an 18-year period at the Mayo Clinic, in which external carotid ligation was done for nasal hemorrhage. In a later report of 212 cases of severe epistaxis, Hallberg[27] mentioned that 17 had required external carotid ligation. Another case of ligation of the external carotid artery for nasal hemorrhage was reported by Fitz-Hugh and Risher.[21]

Ligation of the internal maxillary artery for severe hemorrhage from the sphenopalatine artery was advocated by Seiffert,[42] as being more effective than occlusion of the carotid

artery, which displays anastomoses which may keep the circulation in motion in the bleeding area. His technique included removal of the facial wall of the antrum of Highmore, chiseling out a large section of its posterior wall which was removed with a hook, after which the periosteum was cleft, and the artery isolated near its point of exit from the sphenopalatine foramen, and ligated. Hirsch,[32] in 1936, reported a case in which this approach was used. At about the same time, Sewall[55] mentioned that he had used the transantral approach for ligation of the internal maxillary artery in two instances of profuse bleeding. Kirschner[42] commented that ligation of the internal maxillary artery and other terminal branches of the external carotid is rarely necessary. Although the internal maxillary artery may be the source of profuse bleeding, particularly in some cases of trauma, such hemorrhage is usually dealt with by ligation of the external carotid.

During recent years, there have been several reports of ligation of the anterior ethmoid artery for nasal hemorrhage. Goodyear,[25] who reported a case in 1937, is usually credited with priority in the ligation of the anterior ethmoid for epistaxis, but this was an essential feature of the fronto-ethmosphenoid operation, which was performed with relative frequency in the decades preceding Goodyear's report. Luongo,[46] Sewall,[55] and Simpson[56] all mentioned the ligation of the anterior ethmoid artery (and the other ethmoid arteries) as applicable to the control of obstinate nasal hemorrhage.

The incision of the ethmoid ligation is made along the eyebrow. Luongo described the elevation of the periosteum as being started at the upper third of the incision with downward sweeping movements, followed up by forward ones. The anterior ethmoidal artery is usually found in the orbit within 3 cm. It is tied and severed at the foramen. The posterior ethmoid artery, in case it should be the source of hemorrhage, is found a short distance behind the anterior vessel.

Besides Goodyear, others who have reported ligation of the anterior ethmoid artery for severe epistaxis are Macbeth,[47] Oppenheim, *et al.*,[53] who reported two cases, and Fitz-Hugh and Risher.[21] In the case described by the latter authors, ligation of the external carotid artery failed to control the hemor-

rhage, and the anterior ethmoid also had to be interrupted before the bleeding ceased completely.

The foregoing outline of the instruments, agents and procedures available for the control of nasal hemorrhage serves to demonstrate that there is no one routine or best treatment for this condition, which arises from multiple causes and exhibits a wide range of severity. Each case has to be treated according to the indications presented, and the purpose in every instance is to control the bleeding as promptly as possible with the most conservative means that may prove effective.

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MEETING OF THE GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Georgia Society of Ophthalmology and Otolaryngology will meet March 11-12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia.

The speakers will be: Dr. Francis H. Adler, Philadelphia; J. W. McCall, Cleveland; Dr. J. A. Hilger, St. Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto, Canada. For further details, address ALTON V. HALLUM, M.D., Secretary, 245 Doctors Bldg., Atlanta 3, Ga.

NORMAL HEARING AND ITS RELATION TO AUDIOMETRY.

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INTRODUCTION.

The advantages of audiometry over other methods of examining threshold auditory acuity are such that in many countries the audiometrist is becoming an indispensable aid to the otologist; yet it is clear that much of the advantage is lost unless certain standards can be maintained as to the characteristics of the audiometer and the way it is used. In this country, something has been done in standardizing the instrument,[1,2] though little has been standardized in the way it is used. In many other countries, likewise, national bodies have written specifications for the instrument. So far as the writer is aware, no other country has adopted more detailed specifications than contained in the reference cited above.

Many features of an audiometer must be standardized, but none of more fundamental importance than the frequencies used and the intensity level of the acoustic output which describes "Normal Hearing." There is by now a fairly general use of the octaves from 125 to 8000 cps as the frequencies to be used, but a disagreement exists as to just what intensity, at any particular frequency, shall describe "Normal Hearing."

American audiometers set "Normal Hearing" in accordance with values established by the U. S. Public Health Service and on record in the National Bureau of Standards (NBS).[3] Almost from the time these values were established, however, dissatisfaction has been expressed. Bunch,[5] for example, with probably the most extensive clinical experience in the country, set his own "Normal" at intensities up to 10 db fainter than the present standard at the higher frequencies. It is well known that the average hearing of those in the age group

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20 - 29, tested at the 1939 New York World's Fair[22] was, at 3520 and 7040 cps, up to 10 db *better* than the same age group tested by the Public Health Service Survey of 1935 - 36 —even though the World's Fair study included some ears known to be seriously defective. Lüscher and Zwislocki[11] stated that their American audiometers yielded values, on supposedly normal-hearing individuals, of 10 and 15 db fainter than our American standard. The British[6,26] have recently found thresholds for normal-hearing individuals which they believed to be considerably fainter than our standard. It seems evident that a re-examination of the standard is called for.

Of course, so long as one can calculate from audiometric data an individual's hearing threshold, for the equipment used, in terms of the international reference intensity (10^{-16} watt/cm,^{2} equivalent to a sound pressure level (SPL) of .0002 dyne/cm^2), it is for scientific purposes immaterial just what intensity level is produced by "zero" on the audiometer. For example, it can be calculated that a 20 db hearing loss on an American audiometer is equivalent to a threshold of 37 db above reference SPL in a certain standardized closed coupler, and the same figure could still be derived no matter where the audiometer zero was maintained; however, the reporting of audiograms in terms of threshold SPL does not have the merit of simplicity, and furthermore is quite unnecessary if agreement can be reached internationally on just what "Normal Hearing" (*i.e.*, audiometric zero) should be.

1. History of American "Normal Hearing."

Accounts of the development of the audiometer in this country and abroad have been given by Bunch,[5] and of the fixing of American "Normal Hearing" by Watson and Tolan.[23] Only those features pertinent to the present question will be detailed here.

The first really successful commercial audiometer in this country was the Western Electric 2A. It was calibrated at the Bell Telephone Laboratories, "Normal Hearing" being taken as the average dial setting at threshold of otoscopically normal observers between 20 and 30 years old, the num-

ber variously given as 36[20(p. 276)] or 41.[14(Bull. 4, p. 1)] Earphones used were the W.E. Type 552. Similar equipment was used in the Public Health Survey in 1935-?6. In that survey, careful physical calibration was maintained among the 17 different 2A audiometers, and the 31 audiometricians exercised the correct precautions looking toward reliable data. Subjects were volunteers but, of course, inexperienced in psycho-physical judgments.

Data were averaged from both ears of 1242 persons, aged eight to 76 years, normal by clinical examination, with history of no loss for speech in either ear, and whose air conduction audiograms for both ears did not exceed a variation of 20 db. It is this group, then upon which American "Normal Hearing" depends.[14 (Bull. 4, p. 3)]

The average thresholds at the eight test octaves from 64 to 8192 cps for this group were found in terms of dial settings on the W.E. 2A audiometer. The average threshold voltage to the ear phone at each frequency was found to be as follows:[1]

cps	Db below 1 volt
128	73
256	88
512	102
1024	109
2048	111
4096	107
8192	75

Shortly after this information was available, a much improved earphone was produced, the W.E. Type 705A. Three of the most stable phones of this type were compared with the standard Type 552 units, by loudness balancing using six observers and three loudnesses (threshold, 20, and 40 db over threshold). The voltages at the 705A terminals were thus found which produced the equivalent threshold SPL of the Type 552 phones.

The development of the Type 9A 6cc brass coupler at NBS provided a way to pass from earphone voltage as a standard, to acoustic output in a standard cavity the volume of which has an impedance approximately equal to the equivalent volume of the ear. This includes the volume behind the tym-

panic membrane. The 705A earphone is placed at one end of the cylindrical cavity, and a sensitive microphone, the W.E. 605AA, calibrated in dynes/cm,[2] is placed at the other end. Proper acoustic seals and precautions against vibration are maintained. Threshold voltage is then applied to the terminals (or usually 60 db above threshold voltage), and the SPL in the cavity is read with the microphone. By this technique the NBS is able to state "Normal Hearing" as the acuity of an observer whose threshold is reached when the acoustic output of the W.E. 705A phone is such as to produce the following SPL in the 9A coupler:[2,3]

cps	SPL in db re .0002 dyne/cm^2
125	54.5
250	39.6
500	24.8
1000	16.7
2000	17.0
4000	15.1
8000	20.9

Note: Original data were at octaves of 128; data for octaves of 125 were obtained by graphic interpolation.

The great advantage of this standard is that a laboratory need not actually have on hand one of the three standard 705A earphones, now reposited at the NBS, with which to compare its own equipment, but can utilize a 9A coupler to transfer standards to his own 705A phone, and thence, by loudness-balancing, to any other earphone, or earphone-cushion combination, for which the 9A coupler is appropriate.

Loudness balancing has recently been performed by the NBS[15] between their 705A units and two other types of earphones now in common use with audiometers. From these data has been derived the SPL in the 9A coupler which produces equivalent threshold loudness with the 705A standards:

cps	Permoflux PDR-1 in MX-41/AR Cushion	Permoflux PDR-8, in MX-41/AR	W.E. 705A
125	51.3	52.4	54.5
250	40.4	38.8	39.6
500	25.6	24.5	24.8
1000	17.8	16.5	16.7
2000	17.5	17.1	17.0
4000	11.6	14.5	15.1
8000	28.1	28.3	20.9

2. What is the best method of specifying "Normal Hearing?"

Standardizing "Normal Hearing" in a closed acoustic coupler, as outlined above, is an immensely valuable technique for practical reasons, and it would be all that would ever be required if the coupler perfectly simulated the human ear, but unfortunately the 9A coupler does not perfectly simulate even an average human ear; furthermore, the outputs of the phones commonly used on present-day audiometers vary with variations in the acoustic impedances of individual ears. Thus, a phone which generates a certain SPL in the 9A coupler, may generate different levels when coupled to human ears.

Wiener and Filler[27] dramatically showed this when they impressed constant voltage upon an earphone, and mounted it successively on 11 men. Sound pressure level was measured by a small probe tube inserted under the earphone cushion and led to a microphone. Now if the earphone generated the same SPL for all ears, the measurements would show a negligible variation; but, as a matter of fact, the measurements differed from man to man by as much as 10 db at many frequencies, and up to 20 db at the extremes.

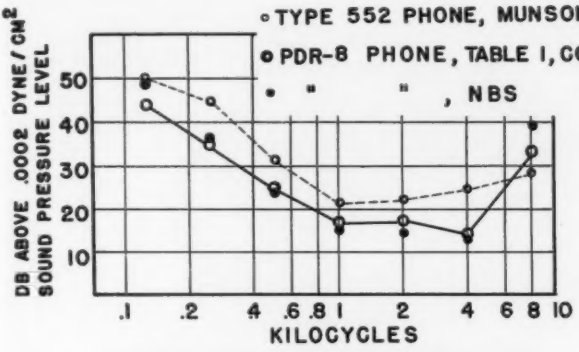
This result illustrates the danger of measuring threshold pressures in a transfer coupler rather than in the ear canal itself: let us take two men, in one of whose canals the earphone is 10 db more efficient than in the other's; at threshold, their earphone voltages (and, therefore, SPL in the 9A coupler) will differ by 10 db, yet in reality their thresholds may be identical, in terms of SPL at the eardrums. Here the threshold by coupler SPL is an erroneous datum. Only by probe technique could the true state of affairs become known.

The practical feasibility of probe tube technique was accentuated by Filler, Ross, and Wiener,[8] who found that results were not affected by "drastic" bending of the flexible tube, by "small" variations in length, or by angle of incidence of sound between 200 to 8000 cps.

The writer concludes that for the careful determination of SPL at an individual's threshold, a direct reading of threshold SPL by probe tube is preferable to an inferred reading with the intermediary of the brass coupler.

3. What is "Normal Hearing" in terms of ear canal SPL?

Standardization groups in this country have not as yet specified "Normal Hearing" in terms of anything but the 9A coupler. It would, however, be of great practical importance if it were possible to state what SPL *measured in the ear canal* actually constitutes a "Normal Hearing" standard. In this way one would avoid all the problems connected with discrepancies between the 9A or any coupler and the human ear,



ESTIMATES OF EAR CANAL SPL AT AMERICAN "NORMAL"

FIG. 1

discrepancies between couplers, the necessity to construct a new coupler for every new type of earphone or earphone-cushion combination, and all the problems concerned with loudness-balancing among earphones, etc.

Munson[20] has provided a bridge by which we may pass from "Normal Hearing" on American audiometers, to SPL in the ear canal. On a "small number of people" he set up "Normal Hearing" on the 2A audiometer and measured SPL by probe "at the opening of the ear canal." In this work, note that the subjects were used only as passive couplers between ear-

phone and microphone. We have, now, only to add to these figures the corrections made to the 2A "Normal" points by the Public Health Survey in order to obtain the SPL at the opening of the ear canal by NBS standards. Munson's data, corrected as described, are in Fig. 1, and represent American "Normal Hearing" in terms of SPL under the 2A ear-

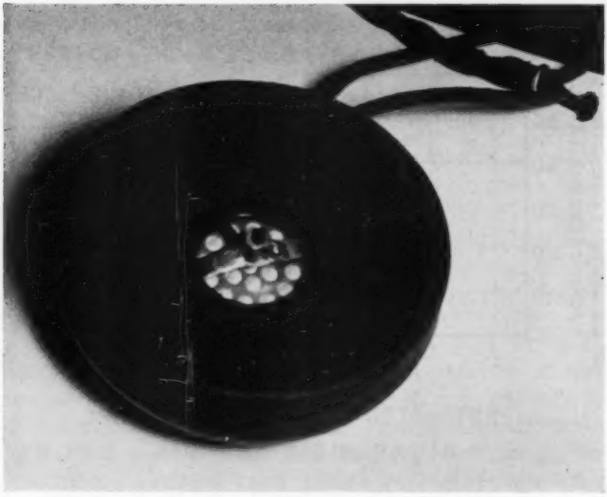


Fig. 2. Experimental Phone, Permoflux PDR-10 with Probe Tube Affixed.

phone (it must be cautioned that these figures are not necessarily correct for other phones or phone-cushion combinations).

Another and rather different estimate of canal SPL at American "Normal" is available in a recent preliminary report[15] from the NBS: using 16 ears as passive couplers just as Munson did, a constant voltage was impressed on the W.E. 705A phone and also on a Permoflux phone, Type PDR-8. For both phones, SPLs developed in the ear canals and (at

the same voltage) in the 9A coupler were reported for headband forces of 1000, 1500, and 2000 grams.

The writer computed the difference in the NBS study between the coupler SPL and the canal SPL, and used this difference in Fig. 1 to estimate canal SPL at American "Normal Hearing." Data for the PDR-8 phone only is entered,

Fig. 3. View of Phone Holder, Probe Tube Microphone, and Sound Pressure Level Measuring Equipment.

the 705A phone yielding very similar though not identical results. At 8000 cps, for example, the difference between the 705A and the PDR-8 earphones amounted to as much as 13 db.

The discrepancies in Fig. 1 between Munson's estimate and that of the NBS led the writer to resolve the problem by still a third estimate. A Permoflux PDR-10 earphone was fitted with an MX-41/AR cushion (see Fig. 2) and mounted in a

plastic helmet (see Fig. 3) with a calibrated pneumatic system providing a force of 2000 grams against the head. A probe tube 8.8 cm long, o.d. 1.9 mm. i.d. 1.2 mm. was fastened to the phone as in Fig. 2. Its tip looked directly into the meatus, and protruded 11 mm from the face of the earphone. It was led to a small brass tube and attached face plate for screwing onto a 640AA microphone exactly as specified by Nichols, *et al.*[16] and as re-published by Beranek.[4]

This system was connected to a calibrated W. E. 640AA microphone and to the Western Electro-Acoustic Company's pre-amplifier, octave-band filter, and voltmeter calibrated in dynes per cm^2.*

TABLE I.

SOUND PRESSURE LEVEL IN EAR CANAL AT AMERICAN "NORMAL HEARING," FOR THE PERMOFLUX PDR-8 PHONE IN THE MX/41/AR COUPLER (Data Taken by Probe Tube under an Experimental Phone, and Transferred to the PDR-8 Phone by Loudness Balancing).

cps	Median	Mean	Standard Deviation	PDR-8 is More Efficient Than Experimental Phone by:
125	44.3	45.5	6.51	3.7 db
250	35.0	33.9	4.47	1.8
500	25.2	25.1	1.63	−0.6
1000	17.2	17.3	1.31	−0.3
2000	17.6	17.1	2.48	0
4000	14.3	13.9	2.68	1.4
8000	33.4	33.6	5.90	−2.9

The probe tube was calibrated by the writer in the 9A coupler, the tube tip entering the cavity in three ways in succession: (a) connected to the earphone as described above, and, therefore, entering the cavity from above; (b) entering the side of the cavity; (c) through a hole in the middle of a brass plug which replaced the microphone at the bottom of the cavity for this measurement. A second independent measurement at all three positions was completed, and a mean of all six readings was computed for each audiometer frequency. The range of variation among the six readings was never as

* The maintenance of this equipment was in the daily charge of Mr. C. E. White, E.E.

much as 4 db at any frequency, and the mean of the two readings at any position never differed by as much as 1 db from the final mean adopted, except at 4000 where it amounted to 1.8 db for one position.

When this equipment was assembled and calibrated, the first 50 men reporting to this laboratory for hearing testing were fitted with the helmet, a force of 2000 grams was applied, and voltage applied to the phone which generated "Normal Hearing" SPL in our 9A coupler. Canal SPL was then read.

The results are in Table I, and the median reading is entered in Fig. 1. Our data are seen to confirm the NBS report and are used in the rest of this paper as an estimate of American "Normal Hearing" in terms of ear canal SPL.

4. Is this inferred canal SPL confirmed by hearing surveys with probe tube technique?

While Table I can be thought of as a standard for canal SPL at "Normal Hearing," it must be remembered that it is based upon the original Public Health Survey, with which some dissatisfaction has often been expressed. What one wishes to know, now, is whether the Public Health Survey definition of "Normal Hearing," as expressed in Table I, agrees with the results of other surveys, by probe tube, of the hearing of clinically normal persons.

Sivian,[19] in 1928, was the first to use a probe tube, inserted about half-way into the meatus to a point 1-1.5 cm from the eardrum. He determined thresholds at frequencies from 500 to 15,000 cps on eight ears, using two procedures: (a) the ear open and stimulated by loudspeaker, and (b) the ear under an earphone, the tones matched in loudness with the free field situation. SPL was measured by probe tube in both cases. Agreement was said to be good. Sivian's thresholds however, are rather less acute than others subsequently reported, and have generally been disregarded as coming from too small a population.

Munson,[19] in 1932, also used the probe tube to study 22 ears from 62 to 15,000 cps. Up to 2500 cps, the tube was inserted just under the earphone cushion; at higher frequen-

cies the earphone calibration was obtained from Sivian's work, at 1-1.5 cm from the eardrum, . . . "on the assumption that equality of loudness of the tones from the loudspeaker and from the receiver, represent equal pressures on the drum." Later work[12,13,17,18] has shown this to be true for the frequencies involved here.

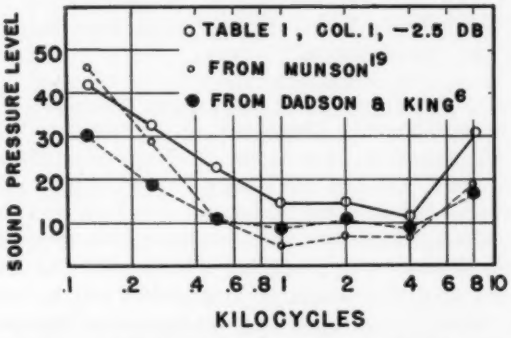
Munson's work is especially significant since it forms the major basis for the curve accepted by the American Standards Association as Minimum Audible Pressure. This curve, taken directly from the paper of Sivian and White[19] is at frequencies above 250 cps largely a slight smoothing of Munson's data (except for a 10 db discrepancy at 12 kc.).

At 250 cps and below, Sivian and White gave greater weight to the work of Wegel, *et al.*[25] who used insert earphones enclosing about 1 cc, and calibrated on a 1 cc. coupler, and the data of Fletcher and Wegel[9] who calibrated their earphones by loudness balance against a thermophone calibrated in a 1 cc. coupler. Reasoning from more recent work on the divergences of coupler SPL from eardrum SPL at lower frequencies, the writer believes that Munson's data form a more nearly correct picture of Minimum Audible Pressure than the composite curve of Sivian and White. Accordingly, Munson's data are entered in Fig. 4 as the best estimate in 1932 of canal SPL for normal hearing.

The next reasonably large surveys of canal SPL for normal hearing were performed by British teams, Dadson and King,[6] and Wheeler and Dickson,[26] Dadson and King have recently found, on 99 otologically normal men and women aged 18-25 years, thresholds which agree substantially with Munson. They regard it as unjustified to include older ages in a study of normal hearing since one thereby deliberately includes the hearing defects which accompany advancing age. There is good reason for this point of view. As they state, "It seems natural to regard the primary function of a pure tone audiometer as being the comparison of the hearing of a subject with a standard representing hearing which is free from impairment, and it thus seems more satisfactory to adopt as a standard of reference a group of subjects whose auditory

state is the most likely to be free from impairment of any kind." (p. 367)

Fig. 4 reproduces their mean of two readings of threshold SPL by probe tube. It can be seen that their data are of the order of magnitude of Munson's data except at 125 and 250 cps, where the British thresholds are respectively about 13 and 10 db fainter.



NOTE: BRITISH DATA FALL 3–14 DB FAINTER THAN AMER. "NORMAL"

COMPARING EAR CANAL SPL AT AMERICAN "NORMAL" WITH TWO PROBE TUBE SURVEYS
FIG. 4

The study of Wheeler and Dickson, using similar equipment and identical calibration facilities, reported closed-coupler data at threshold on 514 men aged 18-23 years, clinically normal, intelligent, and able to pass a whisper and tuning fork test. A clinical audiogram was obtained but was not used to select the population further, and the clinical audiogram was not reported. The technique and cautions used seem without question to have been impeccable. The data reported can be directly compared with Dadson and King's data; not, it is true,

with their canal SPL but with their closed-coupler data which correspond to their canal SPL at threshold. The data of Wheeler and Dickson on this basis differ from those of Dadson and King by only 2 db at the most (mean values at 2000 cps), and usually by much less, and are, therefore, not graphed in Fig. 4. This large survey lends considerable support to the figures for canal SPL at threshold for normal-hearing ears as reported by Dadson and King.

For comparison, American "Normal Hearing" in terms of canal SPL has been entered, minus 2.5 db to allow for the fact that relatively coarse intensity steps were used in the Public Health Survey.

We have collected in Fig. 4, now, three estimates of canal SPL at normal-hearing threshold: *1.* American "Normal Hearing" expressed in those terms, *2.* Munson's 1932 survey of 22 ears, and *3.* Dadson and King's 1952 survey of 198 ears. The differences between the American standard and the two laboratory surveys are clear, and a further difference between the laboratory studies appears, as noted earlier, at 125 and 250 cps. Evidently it would be impossible, on the basis of these data alone, to fix upon any international standards in audiometry.

It seemed in order to conduct another laboratory study in this country along lines of the British work, to help resolve apparent discrepancies. The present paper is one such study.

SUBJECTS.

In this work, the selection and number of subjects can be vital. Our subjects were men 17-23 years of age, physically healthy, with no service-connected auditory trauma, and with an intelligence quotient of 100 or better. None knew of any family-connected hearing disability or had any history of severe ear infections or operations. All were normal by whisper tests. One man only, of a series of 51 consecutive men reporting to this laboratory for routine hearing testing, was rejected from this study on the grounds that he had a hearing loss of more than 15 db at two or more audiometer frequencies. The remaining 50 men were accepted, and data on

their left ears form the basis of this study. Testing was completed within two weeks after starting the study. The assistance of Mr. C. K. Myers, who collected data on nine of the men, is gratefully acknowledged.

EQUIPMENT AND PROCEDURE.

A Maico E-2 audiometer was modified in two ways: *1.* The interrupter circuit was entered and external leads provided which made and broke the circuit by means of a rotating disc, photocell, and relay in such a way that the tone was on for one second (counting from the point at which 100 per cent intensity was reached), and off for one second. *2.* The output of one channel was led to an impedance-matching transformer and to a bank of attenuators in 10, 1, and 0.1 db steps. Another impedance-matching transformer was inserted, and the output finally led to the Permoflux PDR-10 phone in the cushion and helmet shown in Fig. 3.

The subject was seated comfortably, the helmet and earphone adjusted, and 2000 grams force applied. The subject was given instructions to raise his index finger all the while a tone was heard, and drop it otherwise. Practice was given at 500 cps with the intensity moved in 5 db steps down toward threshold. No subject seemed confused by the instructions, or acted inconsistently except as the region of psychophysical indeterminacy was approached.

After practice and an opportunity to ask any question thresholds were collected at the stated audiometer frequencies 125, 250, 500, 1000, 2000, 4000, and 8000 cps.* At each frequency, upon the first sign that the subject was uncertain whether he heard a tone, the intensity was increased 5 db or until clear-cut responses were obtained. At that point, the intensity was decreased in 1 db steps, allowing at least three spurts per level. Threshold was considered to be that intensity setting last heard two of three times. The intensity was then decreased 5 db and an ascending series in 1 db steps begun. Threshold was considered to be at that setting first heard two of three times. Another descending and another

* These frequencies were determined by a Conn Chromatic Stroboscope to be: 125.7, 251.1, 497.9, 988.6, 2045.2, 3978.5, and 7576 cps.

ascending series were given, and the final reading noted down as the mean setting of the four threshold-crossings, to the nearest whole number. Of course, human response being what it is, no audiometrician can hold without any deviation to a rigid plan, but very few liberties were taken with this pattern.

At the conclusion of the seven thresholds, all extraneous attenuation was removed, the audiometer dial was turned to 70 db over zero, and the canal SPL read for each frequency. The audiometer was calibrated to the nearest 0.1 db every day on the 9A coupler; from all this information could be derived the subject's hearing loss with respect to the American closed-coupler standard, and finally the SPL in the ear canal at threshold could be computed.

RESULTS.

The mean and median threshold SPLs, and the standard deviations are given in Table II.

TABLE II.

SOUND PRESSURE LEVEL AT THRESHOLD, MEASURED BY PROBE TUBE UNDER EXPERIMENTAL PHONE, AND TRANSFERRED TO THE PDR-8 PHONE BY LOUDNESS BALANCING.

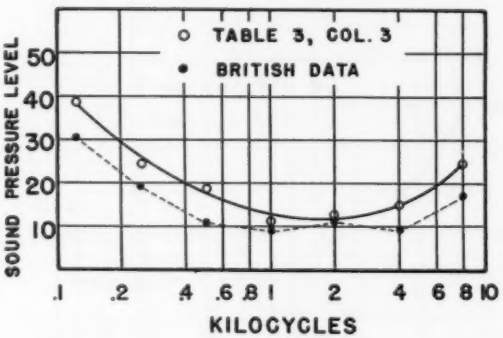
cps	Median	Mean	Standard Deviation	Probe Tube Attenuation
125	38.5	37.9	7.41	−2.4
250	24.4	24.5	5.46	−7.7
500	18.7	18.9	4.77	4.5
1000	11.3	11.9	5.88	8.2
2000	12.4	14.1	7.35	12.4
4000	15.2	17.5	10.90	19.8
8000	25.1	26.4	16.40	27.4

From the shapes of the distributions, as well as from other considerations, the writer concludes that the median is the more useful measure, and it is the median column in Table II which has been plotted in Fig. 5. This graph is our best estimate to date of canal SPL at threshold for normal-hearing ears in this country. It confirms within two or three db (except at 8000 cps) a previous estimate by the writer* on 20

* Unpublished mimeographed memorandum. "Notes on the International Specification of 'Normal Hearing'." U. S. N. Medical Research Laboratory, 1 October, 1953.

ears selected in a manner identical to this study, but with a lighter earphone force and with a somewhat different probe tube.

In this figure also, Dadson and King's data are replotted for easy comparison. The British thresholds are 2-8 db fainter. These differences may be explained in one or more of three



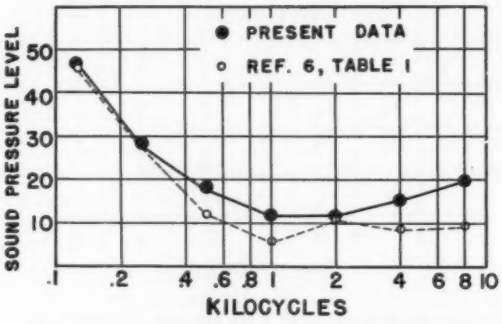
COMPARISON OF PRESENT DATA ON SPL AT THRESHOLD IN EAR CANAL, WITH BRITISH DATA[6]

NOTE: DIFFERENCES MAY BE EQUIPMENTAL

FIG. 5

ways: *1.* Assuming more rigid selection on the part of the British; but this does not seem likely since a somewhat more rigid selection (use of the clinical audiogram) was used for the present data; *2.* The rather amusing assumption that British ears are keener than American, and *3.* Assuming that the difference lies in the two sets of data being dependent upon the specific earphone, earphone-cushion combination, artificial ear or closed coupler, manner of calibrating the probe tube, and perhaps other factors. It seems most likely that

here we will find the sources of discrepancy, for a final adjudication. Already a serious discrepancy has been observed (see our Fig. 1) as to just which canal SPL constitutes American "Normal Hearing," as between the 705A phone and the 552 phone, even though the two units had been balanced in loudness and should, one would suppose, yield the same datum. It can well be imagined that if the British 4026A phone used



COMPARISON OF PRESENT DATA ON SPL AT THRESHOLD IN A CLOSED COUPLER, WITH BRITISH DATA[6]

FIG. 6

by Dadson and King were loudness-balanced with one of the American standard phones, different ear canal pressures might likewise be developed — enough different to account for the divergences between the data of this paper and that of Dadson and King. Only a full exchange of equipment, with data obtained on identical ears, will of course settle this question.

That equipment factors may lie at the basis of the British-American differences is shown by Fig. 7, in which the two

TABLE III.

PROPOSALS FOR A NEW LABORATORY HEARING STANDARD

	A			B		
	BY PROBE TUBE UNDER PDR-8 PHONE			BY 9A COUPLER AND PDR-8 PHONE		
cps	Present Standard (from Table I, Col. 1)	Median Hearing Loss, Present Group	Proposed Standard, 1 db-Step Procedure	Present Standard From Ref. 15 (-2.5 db)	As in Col. (2)	Proposed Standard, Procedure 1 db-Step
125	44.3	-5.8	38.5	49.9	-5.8	44.1
250	35.0	-10.6	24.4	36.3	-10.6	25.7
500	25.2	-6.5	18.7	22	-6.5	15.5
1000	17.2	-5.9	11.3	14	-5.9	8.1
2000	17.6	-5.2	12.4	14.6	-5.2	9.4
4000	14.3	0.9	15.2	12	0.9	12.9
8000	33.4	-8.3	25.1	25.8	-8.3	17.5

populations are compared on the basis of closed-coupler measurements. At two or three frequencies the differences are negligible, and reach a maximum difference at 8000 cps, where coupler measurements are especially dependent upon design factors.

DISCUSSION.

The smooth curve drawn through our data in Fig. 5 can be accepted as a good estimate of Minimum Audible Pressure at the entrance to the ear canal, providing the phone used is a W.E. 705A or one of the Permoflux series, and providing the probe tube is calibrated in the manner described above. The curve should be useful as a standard of young, healthy ears, to any laboratory or other activity interested in, and equipped to measure, Minimum Audible Pressure in this way.

For activities not equipped with a probe tube but with access to a 9A coupler, the smooth curve in Fig. 5 translates into coupler SPL as shown in Table III, Col. 6. For activities with well-calibrated audiometers, but with no direct access to a 9A coupler, it is only necessary to correct the "zero," on the audiometer dial, by the amount shown in Col. 5, Table III, "Hearing Loss." This new zero then is a fair standard to expect a group of young, healthy ears to meet; however, the usefulness in general clinical audiometry of the smooth curve in Fig. 5 rests upon two subsidiary questions: *1.* A decision as to what sort of population audiometric zero should be based upon, and *2.* What allowance should be made, in setting audiometric zero, for the fact that many, if not most, audiograms are collected under conditions far from ideal.

1. What population should audiometric zero be based on?

It seems clear that a basic population should be unselected except that known hearing defects should be eliminated. It seems to the writer that there is no especial reason why one should not accept the definition of the 1242 persons in the Public Health survey as unselected, if one wishes to obtain an average for a very wide range of age, exposure to noise medical history, intelligence, anxiety in a clinic, and a whole host of other factors which might influence threshold data. The only defect is that individuals of advanced age, with a

certainty of some presbyacusis, were included. Unfortunately, the Public Health report never gave the mean threshold for those persons aged 20-29 who formed their fraction of the total 1242 persons on which American "Normal Hearing" rests. If one had this figure, the writer would look no further for an unselected population upon which to base audiometric zero.

A misconception has arisen with respect to the 684 males, aged 20-29, with normal hearing *for speech*, the mean thresholds for which were given by the Public Health report. These 684 men were drawn, not from the 1242 individuals on whom American "Normal Hearing" rests, but from the much larger group of 4662 persons with normal hearing *for speech*, but not necessarily with no audiometric variation greater than 20 db. Of this 4662 group, only 1242, or about 26 per cent, could meet this additional audiometric requirement, and the hearing of the 684 young men was actually worse by 5-6 db at 4096 and 8192 cps, than that of 1242 persons of all ages.

It seems evident, then, that it is not proper to use this group, with known hearing losses, in discussing audiometric zero; nevertheless, it is clear that using only data from the age range 20-29 does not in large surveys improve mean acuity by very much.

Fig. 7 of our previous reference (see footnote*) compares three large surveys in this respect, where the zero reference is the threshold SPL for the ages 20-29, and the curves are the same datum for the age range 10-59 or even more. For the Public Health and San Diego County Fair surveys, differences between the two age ranges are very small, never exceeding 3 db. For the World's Fair survey, on the other hand, differences reach 6-7 db for high tones; but this is perhaps explained by the fact that in that survey the number of individuals used to represent each decade of life was artificially kept approximately constant; there being, for example, more cases at the decade 40-49 than for the decade 10-19. This results in weighting unduly the high-tone losses to be expected in the older groups. Unfortunately, the age distribution of the

* Unpublished mimeographed memorandum. "Notes on the International Specification of 'Normal Hearing'." U. S. N. Medical Research Laboratory, 1 October, 1953.

1242 persons on whom our American standard is based was not reported, so that it is now impossible to re-weight the means for each decade of life; but it seems certain that the large numbers of younger persons in any well-balanced survey make up in good part for the loss of high-tone acuity with advancing age.

We must conclude, then, that the Public Health survey was probably influenced only a very few db by including a wide age range, and that it was otherwise an almost ideal population upon which to base a general audiometric norm.

2. *What allowance should be made, in setting audiometric zero, for the fact that most audiograms are collected under conditions far from ideal?*

If the Public Health population of 1242 is an ideal group, it must be explained why the data from that group as shown in Fig. 4, do not agree with the laboratory surveys of Munson, Dadson and King, and of the present paper. The question has recently been discussed by the British, but the quite correct data bearing on this matter were not adduced. In the first place, it is not correct to use the data for the 684 men of the Public Health Survey, as explained above, but the data for the 1242 individuals. In the second place, it is not quite correct to compare ear canal SPL threshold data from different earphones without correcting for possible differences between the phones as determined by loudness balancing. That this is true is seen from our Fig. 1, where, at equal loudness (namely, threshold as defined by the 1242 individuals), three phones yield quite different data. Munson studied a Western Electric 552 phone, the National Bureau of Standards studied a W.E. 705A phone and a Permoflux PDR-8 (the latter only being plotted here), and this paper studied a converted Permoflux PDR-10 phone. The 705A and the PDR-8 agree well except at 8000 cps, but differ at other frequencies by up to 10 db from the 552.

One correct comparison, the writer feels, is between: *1.* The curve in Fig. 4 which represents canal SPL at threshold for the 1242 individuals if they had been fitted with a PDR-8 minus 2.5 db to correct for the fact that the Public Health

Survey used coarse 5 db steps, and *2*. Our data also in Fig. 5 which represent canal SPL at threshold which would have obtained for 50 individuals if they too had been fitted with a PDR-8.

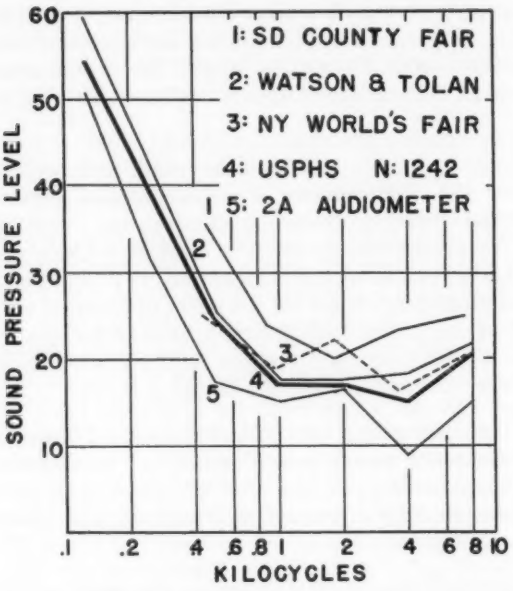
The differences usually cited between the American audiometer and careful probe tube surveys are seen to have been rather sharply reduced. It is clear that, even with the vagaries of audiometricians, differences among audiometers, inexperienced subjects, etc., the Public Health Survey succeeded in coming within 8 db or less of approximating data from careful laboratory studies.

The divergences of up to 8 db are real, and undoubtedly arise from the compromises the psychologist must make in the clinical situation. Many explanations have been offered, the most recurrent being that the clinical subject was untrained. Let us examine this explanation; if it could be established, and numerical values for the effect of training assigned, one could arrive at an audiometric standard for the general population by subtracting from the data for laboratory subjects a correction based upon the training factor.

It was the discrepancy of 15-20 db between Munson's estimate of hearing of audiometer "Normal" of ears aged 20-29, and the Sivian and White curve of MAP, which early attracted notice, and was fully discussed by Steinberg and Gardner.[20] Their conclusion was:

"It is believed that these differences are real and are due principally to differences in experimental techniques. In the Health Survey tests, some 25 or 30 persons per clinic were tested daily. Under these conditions it is unlikely that the operators were able to elicit the lowest threshold responses; also it is unlikely that the subjects, most of whom were being tested for the first time, were in a frame of mind to achieve their best concentration. The data represented by MAP curve were obtained under favorable laboratory conditions, utilizing skilled operators and experienced observers. Even under similar experimental conditions, there must be a considerable difference in the capacity for careful observation between a group chosen at random from the population and a select experienced

group of laboratory observers" . . . "For good ears under favorable conditions, the hearing threshold is represented by the MAP curve; on the other hand, for those in the population who report no difficulty in hearing, the threshold as obtained with



AMERICAN "NORMAL", IN 9A COUPLER, COMPARED WITH FOUR OTHER SURVEYS

FIG. 7

an audiometer in the hands of the otologist is probably better represented by the curves corresponding to the zero setting of the audiometers" (p. 277).

To prove their point, Steinberg and Gardner compared the data of Sivian and White, taken under the best conditions at 1000 cps, with data from 100 inexperienced observers;[21] the

Sivian and White threshold was about 7 db fainter (writer's note: actually, 10 db fainter than Munson's group). This difference was ascribed to care in training and testing the subjects.

The correction of 7 db for practice, rapport, etc., does not wholly explain the discrepancies of up to 12 db, as seen in Fig. 4, between Munson's data and the American standard; moreover, it is likely that the correction is too large. Davis, *et al.*[7] published average "hearing loss" at 500, 1000, and 2000 cps for 21 partially deafened ears by two procedures, a clinical audiogram and a laboratory audiogram in 0.5 db steps. Hearing loss was found to be 57 db by clinical examination, but 65 by laboratory test. The difference of 8 db in favor of the laboratory test is sometimes cited as corroborating the importance of the practice and rapport factors; but a closer look at the data causes the "factors" to vanish altogether. "Hearing Loss" was not referred to the same SPL in the two cases. In the case of the laboratory test, loss was with reference to Sivian and White's MAP, which, averaged at the three frequencies used, equals 9 db re reference pressure. The hearing threshold of these patients by laboratory test, then, was actually $9 + 65 \pm 74$ db. Now the average SPL at these three frequencies by American standards (upon which the clinical examination rests) is 19.5 db. The hearing threshold by clinical test was, then, $19.5 + 57 = 76.5$ db, a matter of only 2.5 worse than the 74 db by laboratory test. And even this discrepancy vanishes if one corrects the clinical audiogram for the coarseness of the 5-db step attenuator.

The almost identical clinical and laboratory audiograms of an earlier paper from this Laboratory* have already been cited. Evidently, without too elaborate precautions, clinical audiograms need not differ markedly from more careful appraisal.

In summing up the evidence on a training factor, the writer concludes that a reasonably careful clinical examination need not appreciably suffer from the subjects' being unfamiliar with audiometry, and that no correction need be added *for*

* Unpublished mimeograph memorandum, "Notes on the International Specification of 'Normal Hearing,'" U. S. N. Medical Research Laboratory, 1 October, 1953.

that reason to the laboratory standards of Table III in order to use them for general audiometry. It is true that the evidence upon which this conclusion rests comes from populations of at least average intelligence (*i.e.*, the clinical patients of Davis, *et al.*, the subjects of the writer's previous study, and the subjects of Wheeler and Dickson); nevertheless any correction for very low intelligence should be made on an individual basis and should not influence the standard itself.

Although training as such can be practically disregarded, there remain many other factors which do actually affect clinical audiograms adversely. Should the standard be adjusted to allow for the irregularities and imprecisions which exist in many clinical situations? The writer feels that the audiometer should be adjusted to the best median performance of young, healthy ears, and that it is unjustified to relax the intensity standard to take account of faulty practice. If there are audiometric workspaces in which no threshold closer than within 15 db of "Normal" can be obtained, or lax audiologists, or audiometers routinely out of calibration, there is no necessity to relax the intensity standards so that those being tested under such conditions are not penalized in some way. It seems more pertinent to seek by education, etc., to close the clinical-laboratory gap rather than to accept a large differential as a necessary evil.

It is true, of course, that the present American standard correctly describes the hearing of large populations. Fig. 7 compares the standard in terms of SPL in 6 cc. couplers with four of the more carefully-done surveys, namely, the original calibrating group for the 2A audiometer, the New York World's Fair group, the San Diego County Fair group, and a group of 100 young adult ears reported by Watson and Tolan. The entries in Fig. 7 were derived as follows:

(*a*) *Public Health Survey.* SPL in the 9A coupler is given directly[2,3] for the 1242 "Normal-hearing" persons.

(*b*) *Original calibrating group for the 2A audiometer.* We know[14] the difference between the 2A "Normal" and the American standard; subtracting this difference gives the 9A coupler SPL for the 2A group as follows:

125	250	500	1000	2000	4000	8000	
54.5	39.6	24.8	16.7	17.0	15.1	20.9	American Standard, 705A phone
3.7	8.0	7.3	1.5	0.6	6.1	5.7	PHS correction to the 2A coupler SPL inferred.
50.8	31.6	17.5	15.2	16.4	9.0	15.2	

(*c*) *New York World's Fair Group.* One can calculate from (*b*) above, by interpolation, the coupler SPL at "Normal" for the 2A calibrating group at the five frequencies used at the World's Fair; Steinberg, *et al.*[22] present a graph showing the db difference between this datum and the reference group (men and women, age 20-29) upon which the World's Fair "Hearing Loss" was based. Subtracting this difference from the coupler SPL for the 2A group, then, gives the coupler SPL for the World's Fair "Normal." Hearing loss of the whole population of the World's Fair group can then be calculated in coupler SPL. This process is shown as follows:

	cps				
	440	880	1760	3520	7040
(1)	20	15.5	16	10	13.5
(2)	+ 0.5	0.0	+ 2.5	+ 0.5	0.0
(3)	20.5	15.5	18.5	10.5	13.5
(4)	+ 2.9	+ 2.9	+ 3.5	+ 6.2	+ 7.1
(5)	23.4	18.4	22.0	16.7	20.6

(1) Interpolated coupler SPL for W.E. 2A calibrating group.

(2) Difference between (1) and the World's Fair "Normal" group (men and women, age 20-29).

(3) Inferred coupler SPL for World's Fair "Normal" group.

(4) Difference between (3) and 35,589 selected ears in the World's Fair group, age 10-59, calculated from Table II, p. 293, ref. 22.

(5) Inferred coupler SPL for total World's Fair group.

(*d*) *The San Diego County Fair Group.* A total of 3482 people aged 10-59, 57 per cent men, were tested binaurally at the same frequencies as at the World's Fair.[24] (A different coupler and a different earphone-cushion combination were used than in the other studies in Fig. 7.) The mean SPLs at threshold for the stated "Normal" group (all 788 men and women aged 20-29) were as follows, compared with the

mean differences (*i.e.*, "Hearing Loss") for the whole group of 3482 individuals:

	440	880	1760	3520	7040
(1)	34	22	18	21	22
(2)	1.1	1.3	2.0	2.5	2.8
(3)	35.1	23.3	20.0	23.5	24.8

(1) Mean threshold SPL in 6 c.c. coupler for "Normal" group.

(2) Difference between (1) and threshold for all 3482 people (calculated from their Table III, p. 481).

(3) Coupler SPL at threshold for all participants aged 10-59.

(*e*) *Watson and Tolan's Group.* These 100 young adult ears were very carefully examined with a commercial audiometer in a soundproof room. No history of hearing defect was included, or any subject with any loss of more than 15 db at any frequency. Subjects were naive. This group, then, was selected by very much the same criteria as the group chosen by the Public Health survey to represent "Normal" except that Watson and Tolan's subjects were all young. They reported mean threshold in terms of the American standard, from which the threshold coupler SPL can easily be computed.

A really detailed comparison of these surveys would be out of place here, but a comment must be made bearing on the question, how well is the present American standard substantiated by other surveys.

It is clear from Fig. 7 that the standard is not too lenient, if one reasons from the fact that large-scale surveys do rather well substantiate it. The World's Fair group approximated the American standard within 1 - 3 db except at 1760 cps, where the Fair population had a *loss* of about 5 db. The San Diego County Fair group yielded mean thresholds with hearing *losses* of 3 - 8 db by American standards, and, of course, the Public Health survey itself, considering all 9324 ears of all ages normal for speech, yielded thresholds which agreed with the American standard within less than 1 db, except at 3520 and 7040 cps, where hearing *losses* of 7 - 9 db occurred.

Had the American standard been too lenient, it would have

occurred that the survey data would show not *losses*, but the reverse.

Evidently, then, the American standard is an approximately correct statement of the hearing of large populations unselected for age, previous hearing history, etc., but this is not to say that these large populations, with known hearing defects, should serve to characterize a standard for unimpaired hearing.

In the matter of setting standards, it is necessary to balance one good against another. To keep the present standard is to maintain the rehabilitation load as we know it today; to adopt a standard such as in our Table III would have the effect of earlier identification of defects. A child with a loss of, say, 5 db at 500 cps is now thought to be perfectly normal within limits of experimental error, but upon adopting the standards in Table III, that child would seem in reality to have a loss of more than 10 db, and to warrant immediate close observation. If many children exhibited such losses, then the workspace, or the technique, or the equipment would be laid under suspicion; thus a general improvement in audiometry would result.

In the writer's opinion, these two benefits outweigh rather heavily the advantage of retaining the present standard, which has only the effects of condoning laxity and of deferring (at the expense of the patient's well-being) the initiation of therapy.

SUMMARY.

The history is briefly reviewed of how American "Normal Hearing" came to be standardized. It was shown that the standard could be specified in terms of sound pressure level in the ear canal as well as in a brass coupler as at present.

The discrepancy usually cited as excessive between hearing norms as collected by large clinical surveys and by laboratory studies is shown to be sharply reduced when the quite correct comparisons are made. Nevertheless, an appreciable discrepancy, though not exceeding 8 db, exists.

It is reasoned that the clinical-laboratory discrepancy is

not due to selection of subjects, nor of familiarity of subjects with psychoacoustic judgments, but resides in features of the clinical situation which are correctable.

A laboratory standard for audiometer "Normal Hearing" is proposed which would more nearly correctly describe the status of an individual's hearing ability in comparison with young, healthy ears, and which would tend at the same time to have a salutary effect upon audiometric practice.

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POST GRADUATE SESSION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A Post Graduate Session in Ophthalmology and Otolaryngology sponsored by the Virginia Society of Ophthalmology and Otolaryngology will meet at the University of Virginia from November 30 to December 3, 1954.

November 30 and December 1, will be devoted to Otolaryngology lectures and December 2 and 3, will be devoted to Ophthalmological subjects. Those interested please contact Dr. Edwin Burton or Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Va.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC., MEETINGS.

MID-WINTER.

Eastern Section Meeting.....Friday, January 7, 1955
Philadelphia, Pa., Hotel Warwick

Triological Council Meeting.....Saturday, January 8, 1955
New York City, The Waldorf-Astoria

Western Section Meeting...Saturday-Sunday, Jan. 15-16, 1955
Los Angeles, Cal., The Town House

Southern Section Meeting.....Saturday, January 22, 1955
Charlottesville, Va., Medical School Auditorium

Middle Section Meeting.....Monday, January 24, 1955
Detroit, Mich., Sheraton-Cadillac Hotel

SPRING.

American Board of Otolaryngology
Richmond, Va., March 6-10, 1955

All Society Meetings will be held at the Hollywood Beach Hotel, Hollywood, Florida.

American Laryngological Association.....March 13, 14, 1955
Triological Society.....(mornings only) March 15, 16, 17, 1955
American Broncho-Esophagological Association
(afternoons only) March 15, 16, 1955
American Otological Society, Inc.....March 17, 18, 1955

The Hollywood Beach Hotel (American plan) is a delightful place to hold the Spring Meetings — you may wish to bring the family along. The Hotel offers its guests such features as ocean bathing, deep sea fishing, 18-hole golf course and country club, tennis and many other activities.

For hotel reservations please communicate with Mr. John W. Tyler, Manager, Hollywood Beach Hotel, Hollywood, Florida.

TWENTY-FOURTH ANNUAL MID-WINTER CLINICAL CONVENTION IN OPHTHALMOLOGY AND OTOLARYNGOLOGY, JANUARY 17 THROUGH JANUARY 28, 1955.

The Research Study Club of Los Angeles announces its Twenty-fourth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 17 through January 28, 1955. For the Ear-Nose-Throat, the main Guest Lecturers will include Dr. Theodore E. Walsh, Professor and Head of the Department of Otolaryngology, Washington University, St. Louis; and Dr. John J. Conley, Chief of the Head and Neck Service, St. Vincent's Hospital, New York City, and Clinical Professor of Otorhinolaryngology, New York University, and other appointments.

Dr. Walsh has long been known to us not only as a professor, but also as a lecturer in our national societies, with particular interest in otologic and rhinologic problems.

Dr. Conley has done a great deal of work in tumors and plastic surgery of the head and neck, as well as being a well trained man in the broader field of our specialty.

Also serving on our program with Didactic Lectures will be Dr. Kenneth C. Brandenburg of Long Beach, Dr. J. C. Almy Harding of San Diego, and Dr. Paul J. Moses of San Francisco.

Instruction Courses in Otolaryngology will be provided by Dr. E. R. V. Anderson, Dr. Norman Jesberg, Dr. Harold Owens, and Dr. Manuel R. Wexler, all of Los Angeles, and Dr. Leo Shahinian of San Francisco.

The main Guest Lecturers for the Eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold, both from the University of Pennsylvania Graduate School of Ophthalmology.

The first week will be devoted to the Ear-Nose-Throat—from Monday to Saturday, January 17 through January 22. The Eye week will cover Monday to Friday, January 24 through January 28. Those of us who confine our work to only one of these specialties may complete either subject in one week.

On Tuesday evening, January 18, the Annual Banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of that week. This will be held at 7:00 p.m. in the Ball Room of the Elks Club.

On Wednesday afternoon of each week motion pictures will be shown, Ear-Nose-Throat subjects the first week, and Eye subjects the second week. These afternoons will also afford time for relaxation, sight-seeing, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting at The Town House immediately preceding this Convention, Saturday and Sunday, January 15 and 16. On Saturday afternoon the scientific session is open to members and all interested otolaryngologists. Saturday evening there will be a dinner meeting for members of the Triologic Society only. Sunday morning, the second scientific session is open to all interested. The president of the Society, Dr. Kenneth M. Day, after attending this Society's Annual Meeting, will be with us during a portion of the Mid-Winter Course of the Research Study Club.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. H. M. Nickerson, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail.

Each applicant must be a member, in good standing, of the American Medical Association *in Order to Become Eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. We are informed that these dues are an income-tax deductible item, as they represent an annual membership fee. Make your Check payable to "Mid-Winter Clinical Convention," and mail to Pierre Violé, M.D., Treasurer, 1930 Wilshire Boulevard, Los Angeles 57. If anything prevents your attendance, this fee will be returned to you.

UNIVERSITY OF FLORIDA MID-WINTER SEMINAR.

The Ninth Annual University of Florida Mid-winter Seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach, the week of January 17, 1955. The lectures on Ophthalmology will be presented on January 17, 18, and 19, and those on Otolaryngology on January 20, 21, and 22. A mid-week feature will be the Mid-winter Convention of the Florida Society of Ophthalmology and Otolaryngology on Wednesday afternoon, January 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p.m., on Wednesday. The Seminar schedule permits ample time for recreation.

The Seminar lecturers on Ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis; and Dr. Milton L. Berliner, New York. Those lecturing on Otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, St. Louis, and Dr. David DeWeese, Portland, Oregon.

ANNOUNCEMENT.

An introductory course in Reconstructive Surgery of the Septum and External Nasal Pyramid will be given, under the direction of Dr. Maurice Cottle, March 19 through 26, 1955, at the University of Oregon Medical School. This will be sponsored by the Department of Otolaryngology.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

November 1, 1954.

Acousticon Models A-17, A-180, A-185 and A-1530.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HE ARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer. Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1. Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes. Microtone Model Skylark; 3 transistors.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill

Paravox Model K.M. (TRANSonic); 3 transistors.

Manufacturer: Paravox, Inc., 2056 East 54th St., Cleveland, Ohio.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex Hearing Aids (three types).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17 (afternoon), 18 (forenoon and afternoon), 19 (forenoon), 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

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Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Eastern Section Meeting, Friday, January 7, 1955, Philadelphia, Pa., Hotel Warwick.
Triological Council Meeting, Saturday, January 8, 1955, New York City, The Waldorf-Astoria.
Western Section Meeting, Saturday - Sunday, January 15 - 16, 1955, Los Angeles, Cal., The Town House.
Southern Section Meeting, Saturday, January 22, 1955, Charlottesville, Va., Medical School Auditorium.
Middle Section Meeting, Monday, January 24, 1955, Detroit, Mich., Sheraton-Cadillac Hotel.
Meeting: Hollywood Hotel, Hollywood, Fla., March 15-16-17, 1955, morning only.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. F. W. Davison, Danville, Pa.
Vice-Chairman: Dr. Guy L. Boyden, Portland, Ore.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

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President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October 9-15, 1955.

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Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Palmer House, Chicago, Ill., October, 1955.

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Meeting: Hollywood Beach Hotel, Hollywood, Fla., March 15-16, 1955, afternoon only.

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Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

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Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Meeting: General Oglethorpe Hotel, Savannah, Ga., March 11-12, 1955.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George McKenzie, Dupont Medical Bldg., Miami, Fla.
Vice-President: Dr. Maurice Edelman, 605 Lincoln Road, Miami Beach, Florida.
Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.
Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

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Vice-President: Dr. Henry B. Perlman, 950 East 59th St., Chicago, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

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Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 9, 1955.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

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Meeting:

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Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.
Spring Meeting: Natural Bridge Hotel, Natural Bridge, Va., May 7-8, 1955.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.
Meeting:

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Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

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Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

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Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

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Meeting:

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Meeting: Bigwin Inn, Lake of Bays, Muskoka, Ontario. June 16 to June 18, 1955.

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Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

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Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

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President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

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